*A Dissertation on*

# *A DESCRIPTIVE STUDY ON OUTCOMES & EFFICACY IN MANAGING ACUTE CHOLANGITIS AND ACUTE CHOLECYSTITIS BASED ON TOKYO GUIDELINES 2013*

*Submitted to*

**THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY**
**CHENNAI – 600032**

In partial fulfilment of the Regulations
for the Award of the Degree of

**M.S. (GENERAL SURGERY)**

**BRANCH - I**


**DEPARTMENT OF GENERAL SURGERY**
**STANLEY MEDICAL COLLEGE**
**CHENNAI – 600 001**

**APRIL 2017**

# DECLARATION

I ***Dr. S.SIVAPRAGASH*** solemnly declare that this dissertation titled **"*A DESCRIPTIVE STUDY ON OUTCOMES & EFFICACY IN MANAGING ACUTE CHOLANGITIS AND ACUTE CHOLECYSTITIS BASED ON TOKYO GUIDELINES 2013 (TG 13).*"** is a bonafide work done by me in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of my chief **Prof. Dr. J.LALITH KUMAR** and my Head of the Department **Prof. Dr. D.NAGARAJAN**

This dissertation is submitted to the Tamilnadu Dr MGR Medical university, Chennai in partial fulfilment of the university regulations for the award of M.S.degree (General Surgery), branch – 1 examination to be held in APRIL 2017.

**SEPTEMBER 2016** **Dr. S.SIVAPRAGASH**

**CHENNAI**

# CERTIFICATE

This is to certify that the dissertation entitled "***A DESCRIPTIVE STUDY ON OUTCOMES & EFFICACY IN MANAGING ACUTE CHOLANGITIS AND ACUTE CHOLECYSTITIS BASED ON TOKYO GUIDELINES 2013***" is a bona fide work done by **Dr. S.SIVAPRAGASH**, post graduate ( 2014-2017) in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfilment of the regulations of the **TAMILNADU Dr. MGR MEDICAL UNIVERSITY** Chennai for the award of **M.S degree (General surgery)** Branch-1 examination to be held in APRIL 2017.

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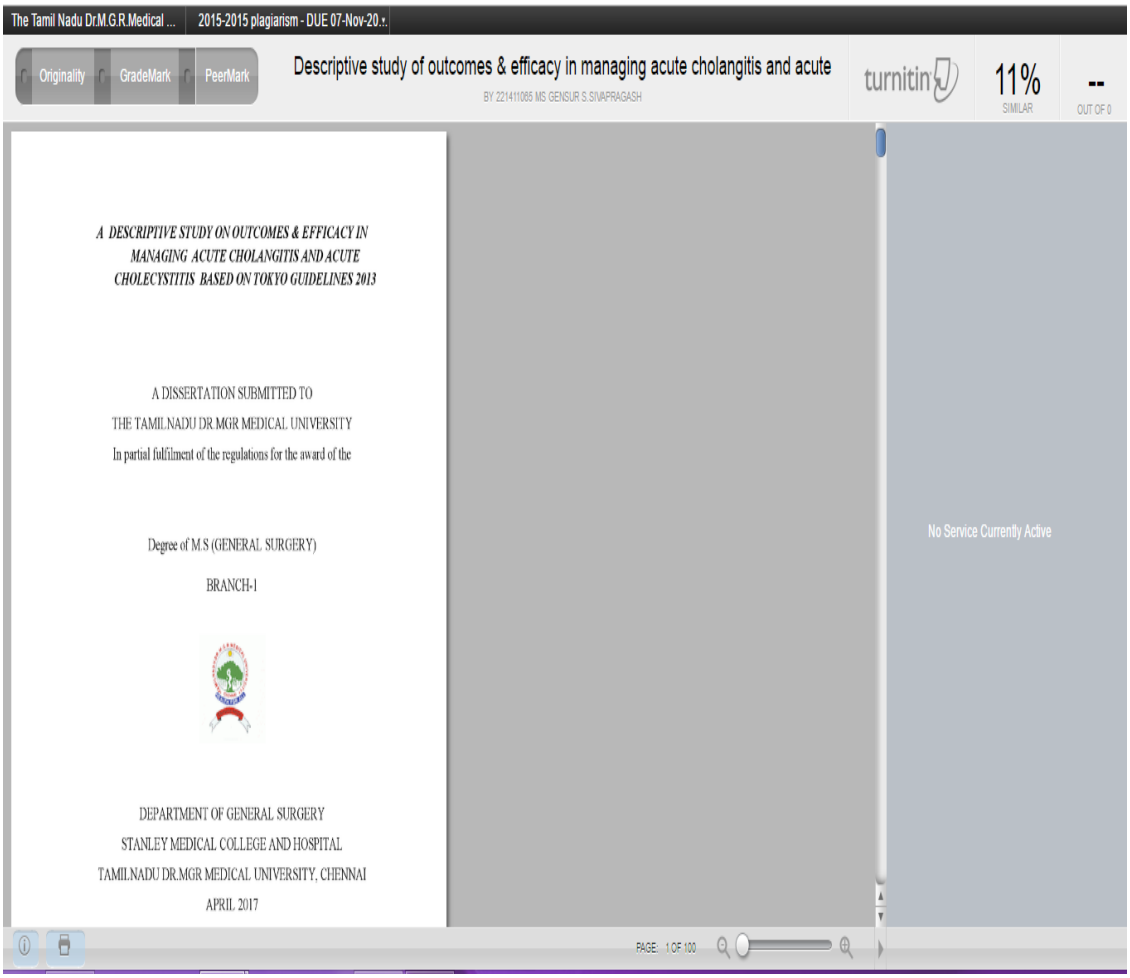
# PLAGIARISM CERTIFICATE



INSTITUTIONAL ETHICAL COMMITTEE,
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| | | |
|---|---|---|
| Title of the Work | : | Descriptive study of outcomes & efficacy in Managing acute cholangitis and acute cholecystitis based on Tokyo guidelines 2013(TG 13). |
| Principal Investigator | : | Dr. S. Sivapragash |
| Designation | : | PG MS (General Surgery) |
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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 14.06.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
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MEMBER SECRETARY, 23/9/16.
IEC, SMC, CHENNAI

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# ABSTRACT

**Background:**

Acute biliary sepsis which includes Acute cholecystitis and Acute Cholangitis are life threatening surgical emergencies which needs early recognition and prompt institution of appropriate management strategies.Except for some well known clinical signs like Murphy's sign and Charcot's triad there are no well defined criteria for the diagnosis of above said biliary infections.Tokyo Guidelines 2013 is expert panel formulated Diagnostic and severity assessment criteria guidelines is the only guidelines available till date which aids in the diagnosis and management of the same.More number of patients can be diagnosed at an early stage of infection and appropriate treatment instituted at the earliest by using this guidelines ,thus avoiding any foreseen complications.In this study,we have assessed the feasibility of using TG 13 guidelines in diagnosing and managing acute biliary infections in Stanley medical college.

**OBJECTIVE**:

This study sought to analyse the outcome of managing acute biliary sepsis And comparing it in lines with TG 13 Guidelines.

**MAIN OUTCOME MEASURES:**

The Pattern of presentation,clinical features and contributing factors of Acute cholecystitis and Acute Cholangitis.

**STUDY DESIGN**: Descriptive study.

**SETTING:** Department of General surgery
Govt. Stanley Hospital ( GSH ) a tertiary hospital in TAMILNADU.

**STUDY DURATION**: JUNE 2016 TO SEPTEMBER 2016.

# METHODS AND MATERIALS

Patients who had been diagnosed as a case of acute Cholecystitis and acute cholangitis were followed up and assesed using the TG 13 guidelines criteria and the outcomes were analysed.

The efficacy of Using the guidelines in our setup and reproducibility of guideline demanding parameters were analysed that were appropriate in our setup.

Clinical details and lab and imaging findings were recorded using a well detailed clinical proforma that included detailed description of analysis of a case of acute cholecystitis and cholangitis. Also other contributing factors,clinical patterns ,presentation ,treatment outcomes were analysed in detail.

Data analysis and master charting was done using Microsoft Excel software

And the results interpreted.

## RESULTS:

Acute Cholecystitis with most common etiology being gallstone disease is more common in males (56%),While Acute cholangitis is more common among elderly females.(58%).> 52 % patients diagnosed with Acute cholecystitis presented to us only after >72 hours of symptom onset. 43 % of cholecystitis and > 50% cholangitis patients were Diabetics Under treatment for the same. Diabetic Males more than 50 years of age with duration of diabetes less than 5 years had increased incidence of cholecystitis and present with a higher grade.

Keywords : Tokyo guidelines, acute biliary sepsis, diagnostic criteria
Early cholecystectomy

.

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**LIST OF ABBREVIATIONS:**

TG 13- TOKYO GUIDELINES 2013

EC - EARLY CHOLECYSTECTOMY

DC - DELAYED CHOLECYSTECTOMY

# INTRODUCTION

# INTRODUCTION

- Acute cholangitis and Acute cholecystitis require appropriate treatment in the acute phase. Severe acute cholangitis may result in early Mortality and Morbidity if appropriate medical care is Delayed in the acute phase.

- Before the publication of the Tokyo Guidelines for the management of acute cholangitis and cholecystitis (TG07) in January 2007 ,which consequently was updated in 2013 there were no practical guidelines throughout the world primarily targeting the treatment of acute cholangitis and cholecystitis.

- TG13 Diagnostic Criteria of Acute Cholangitis and Cholecystitis are criteria to establish the diagnosis when cholestasis and inflammation based on clinical signs or blood tests in addition to biliary manifestations based on imaging are present.

- TG13 guidelines also encompasses a severity assessment criteria based on which patients can be divided into groups which are then managed accordingly as per the severity criteria.

- It allows for early diagnosis and easy management of patients and to determine the necessity for early biliary drainage in case of cholangitis and early cholecystectomy in case of cholecystitis which are vital life saving measures in view of these dreaded biliary tract infections.

# AIM OF THE STUDY

## AIMS AND OBJECTIVES

- To study on the clinical patterns ,presentation of Acute Biliary Infections @ stanley medical college.

- To assess feasibility of application & the efficacy of TG-13 guidelines criteria and norms in our setup.

- To analyse the outcomes of Current treatment trends among the study population in our setup.

# REVIEW OF LITERATURE

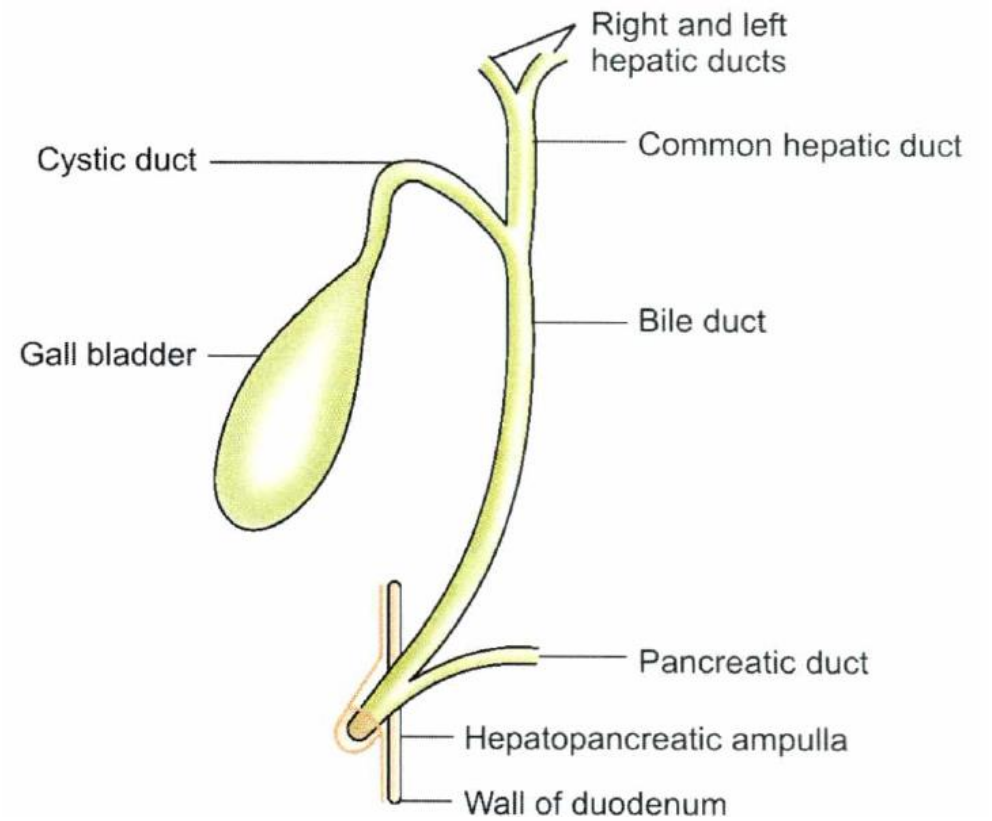
# REVIEW OF LITERATURE

## EMBRYOLOGY OF BILIARY TRACT:

During the fourth week of gestation. A bud arises off the ventral wall of the primitive foregut,which eventually forms the duodenum.The liver diverticulum initially breaks into cranial and caudal portions-the cranial portion becoming the intrahepatic bile ducts and the caudal portion forming the gallbladder and the cystic duct.

By the fifth week of intrauterine life,the cells between the liver bud and the remaining foregut proliferate to form a primitive bile duct**.** The common bile duct becomes occluded with epithelial cell Proliferation as it elongates and by the end of $5^{th}$ week it recanalises moving distally towards the gallbladder which remains solid until $12^{th}$ week .Failure to recanalize is attributed to the pathogenesis of biliary atresia.

## ANATOMY OF BILIARY TREE:



## COMPONENTS OF THE BILIARY TREE :

1. **RIGHT AND LEFT HEPATIC DUCTS**
2. **COMMON HEPATIC DUCT**
3. **GALL BLADDER**
4. **CYSTIC DUCT**
5. **COMMON BILE DUCT**
6. **AMPULLARY REGION**

## GALL BLADDER :

The gallbladder is situated on the undersurface of the anterior inferior sector (Segment V) of the right lobe of the liver. Though often densely adherent, it is separated from the liver parenchyma by the cystic plate, a layer of connective tissue arising from **Glisson's capsule** and in continuity with the hilar plate at the base of Segment IV.

## Dimensions and Capacity

The gall bladder is 7 to 10 cm long, 3 cm broad at its widest part, and about 30 to 50 ml in capacity.

## Parts

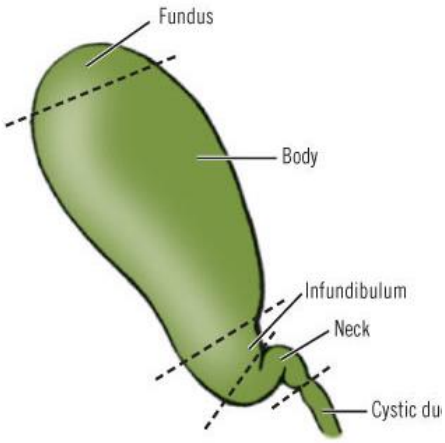
The gall bladder is divided into:

1. The fundus,

2. The body, and

3. The Infundibulum

4. The Neck




Variations in gallbladder anatomy are rare.

These variations include :

(a) bilobed or double gallbladders,

(b) septated gallbladders, or

(c) gallbladder diverticula

The *fundus* projects beyond the inferior border of the liver, in the angle between the lateral border of the right rectus abdominis and the ninth costal cartilage. It is entirely surrounded by peritoneum, and is related anteriorly to the anterior abdominal wall, and posteriorly to the beginning of the transverse colon.

The *body* lies in the fossa for the gall bladder on the liver. The upper narrow end of the body is continuous with the neck at the right end of the porta hepatis. The superior surface of the body is devoid of peritoneum, and is adherent to the liver. The inferior surface is covered with peritoneum, and is related to the beginning of the transverse colon and to the first and second parts of the duodenum.

The *neck* is the narrow upper end of the gall bladder. It is situated near the right end of the porta hepatis. It is continuous with the cystic duct. Its junction with the cystic duct is marked by a constriction. The mucous membrane of the neck is folded spirally to prevent any obstruction to the inflow or outflow of bile. The posteromedial wall of the neck is dilated outwards to form a pouch called the Hartmann's pouch which is directed downwards and backwards. Gall stones may lodge in this pouch.

## CYSTIC DUCT:

The cystic duct arises from the infundibulum of the gallbladder and runs medial and inferior to join the common hepatic duct. The cystic duct is typically 1-3 mm in diameter and can range from 1 mm to 6 cm in length depending upon its union with the common hepatic duct. The mucous membrane of the cystic duct forms a series of 5 to 12 crescentic folds, arranged spirally to form the so-called *spiral value* of Heister. This is not a true valve.

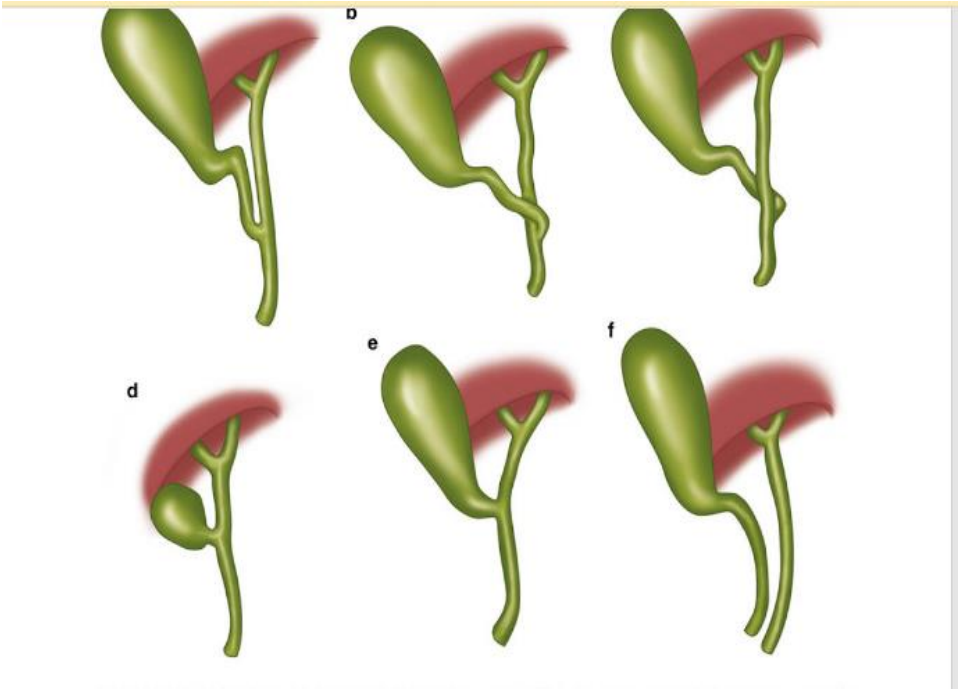
Cystic duct abnormalities are uncommon and include

a) double cystic ducts (very rare),

b) aberrant cystic duct entry sites, and

c) aberrant cystic duct union with the common hepatic duct.

Aberrant entry points for the cystic duct include a low entry into the common hepatic duct retroduodenal or retropancreatic and anomalous entry into the main right hepatic duct or sectoral duct.




## Common Bile Duct :

Bile duct is formed by the union of the cystic duct and

common hepatic duct near the porta hepatis.

It is 8 cm long and has a diameter of about 6 mm.

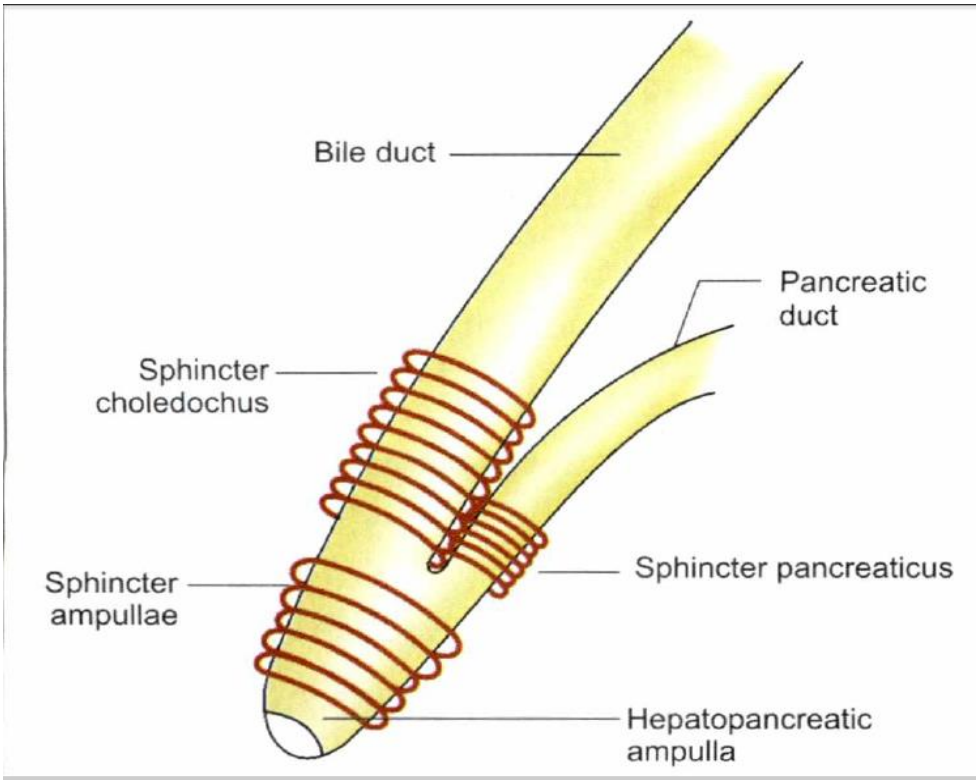
## Course :

The bile duct runs downwards and backwards, first in the free margin of the lesser omentum, *supraduodenal part;* then behind the first part of the duodenum the *retroduodenal part;* and lastly behind, or embedded in, the head of pancreas *infraduodenal part.* Near the middle of the left side of the

second part of the duodenum it comes in contact with the pancreatic duct and accompanies it through the wall of the duodenum, the *intraduodenalpart.* The course of the duct through the duodenal wall is very oblique.Within the wall the two ducts usually unite to form the *hepatopancreatic ampulla,* or *ampulla of Vater.*

The distal constricted end of the ampulla opens at the summit of the major duodenal papilla 8 to 10 cm distal to the pylorus.

## AMPULLARY REGION



The terminal part of the bile duct is surrounded just above its junction with the pancreatic duct by a ring of smooth muscle that forms the *sphincter choledochus (choledochus* = bile duct). This sphincter is always present. It normally keeps the lower end of the bile duct closed (Fig. 22.7). As a result, bile formed in the liver keeps accumulating in the gallbladder and also undergoes considerable concentration. When food enters the duodenum, specially a fatty meal, the sphincter opens and bile stored in the gall bladder is poured into the duodenum. Another less developed sphincter, which is usually but not always present around the terminal part of the pancreatic duct is the *sphincter pancreaticus.* A third sphincter surrounds the hepatopancreatic ampulla and is called the *sphincter ampullae* or sphincter of Oddi.

**BLOOD SUPPLY OF THE BILIARY TREE :**

The cystic artery is the chief source of the blood supply, and is distributed to the gall bladder, the cystic duct, the hepatic ducts and the upper part of the bile duct.Several branches from the posterior superior pancreaticoduodenal artery supply the lower part of the bile duct.The right hepatic artery forms a minor source of blood supply to the middle part of the bile duct.The *cystic artery* usually arises from the right hepatic artery, passes behind the common hepatic and cystic ducts, and reaches the upper surface of the neck of the gall bladder, where it divides into superficial and deep branches.

## FUNCTIONS OF GALLBLADDER :

- ✓ Storage of bile, and its release into the duodenum when required.

- ✓ Absorption of water, and concentration of bile.

- ✓ The normal gall bladder also absorbs small amounts of a loose bile salt-cholesterol compound. When the gall bladder is inflamed, the concentration function becomes abnormal and the bile salts alone are absorbed leaving cholesterol behind.

- ✓ It regulates pressure in the biliary system by appropriate dilatation or contraction. Thus the normal, choledochoduodenal mechanism is maintained.

## PHYSIOLOGY OF BILE:

Volume : 800 to 1,200 mL/day

Reaction : Alkaline

pH : 8 to 8.6

Specific gravity : 1.010 to 1.011

Color : Golden yellow or green.

## COMPOSITION OF BILE

Bile contains 97.6% of water and 2.4% of solids. Solids include organic and inorganic substances.

## SECRETION OF BILE

Bile is secreted by hepatocytes. The initial bile secreted by hepatocytes contains large quantity of bile acids, bile pigments, cholesterol, lecithin and fatty acids. From hepatocytes, bile is released into canaliculi. From here, it passes through small ducts and hepatic ducts and reaches the common hepatic duct. From common hepatic duct, bile is diverted either directly into the intestine or into the gallbladder. Sodium, bicarbonate and water are added to bile when it passes through the ducts. These substances are secreted by the epithelial cells of the ducts. Addition of sodium, bicarbonate and water increases the total quantity of bile.

## STORAGE OF BILE

Most of the bile from liver enters the gallbladder, where it is stored. It is released from gallbladder into the intestine whenever it is required. When bile is stored in gallbladder it undergoes many changes both in quality and quantity such as:

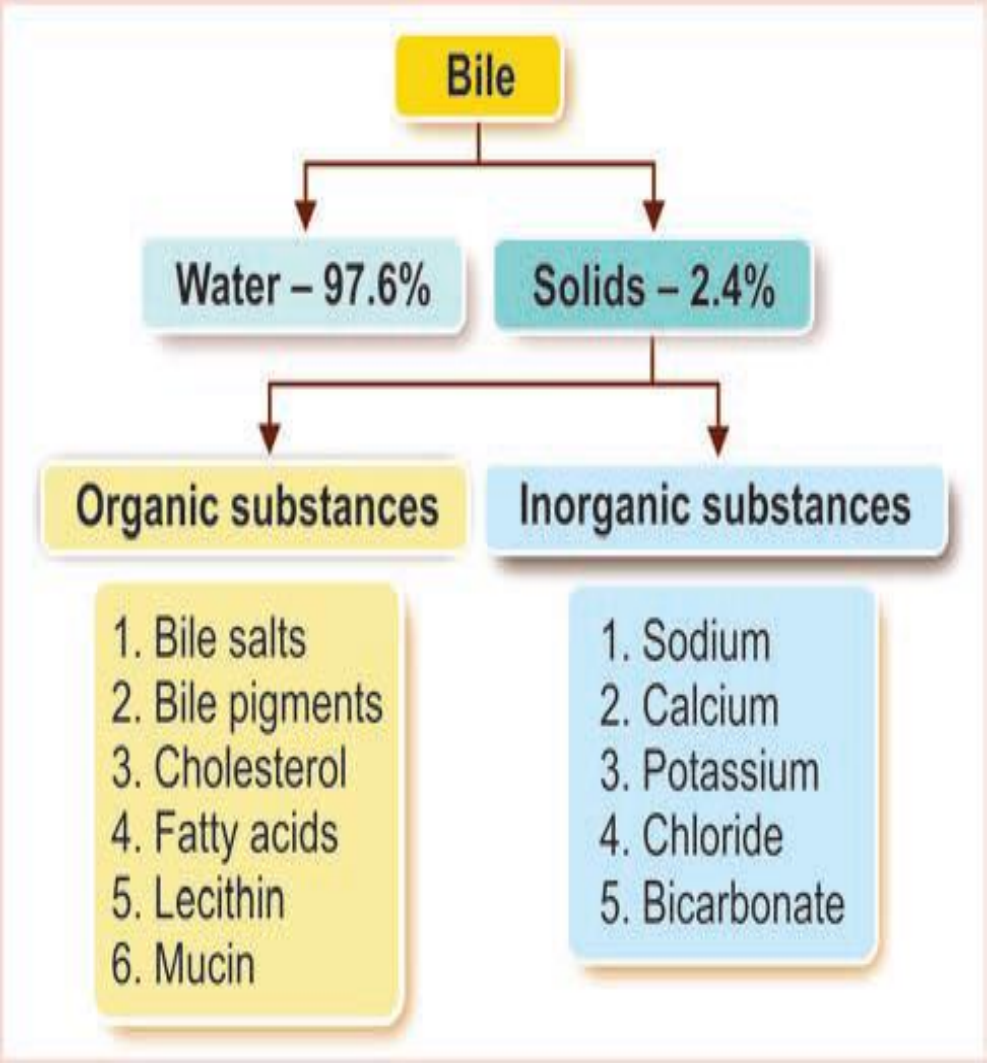
1. Volume is decreased because of absorption of a large amount of water and electrolytes (except calcium and potassium)

2. Concentration of bile salts, bile pigments, cholesterol, fatty acids and lecithin is increased because of absorption of water and electrolytes.

3. The pH is decreased slightly.

4. Specific gravity is increased.

5. Mucin is added to bile.


Bile
Water – 97.6%
Solids – 2.4%
Organic substances
Inorganic substances
1. Bile salts
2. Bile pigments
3. Cholesterol
4. Fatty acids
5. Lecithin
6. Mucin
1. Sodium
2. Calcium
3. Potassium
4. Chloride
5. Bicarbonate

## REGULATION OF BILE SECRETION:

Bile secretion is a continuous process though the amount is less during fasting. It starts increasing after meals and continues for three hours. Secretion of bile from liver and release of bile from the gallbladder are influenced by some chemical factors, which are categorized into three groups:

1. Choleretics
2. Cholagogue
3. Hydrocholeretic agents.

- ✓ **Choleretics**

  Substances which increase the secretion of bile from liver are known as choleretics.

  Effective choleretic agents are:

  i. Acetylcholine

  ii. Secretin

  iii. Cholecystokinin

  iv. Acid chyme in intestine

  v. Bile salts.

- ✓ **Cholagogues**

  Cholagogue is an agent which increases the release of bile into the intestine by contracting gallbladder

  Common cholagogues are:

i. Bile salts

ii. Calcium

iii. Fatty acids

iv. Amino acids

v. Inorganic acids

All these substances stimulate the secretion of cholecystokinin, which in turn causes contraction of gallbladder and flow of bile into the intestine.

## ✓ Hydrocholeretic Agents

Hydrocholeretic agent is a substance which causes the secretion of bile from liver, with large amount of water and less amount of solids. Hydrochloric acid is a hydrocholeretic agent.

# PATHOPHYSIOLOGY OF GALLSTONES:

Definitions

Gallstone is a solid crystal deposit that is formed by cholesterol, calcium ions and bile pigments in the gallbladder or bile duct. **Cholelithiasis** is the presence of gallstones in gallbladder. **Choledocholithiasis** is the presence of gallstones in the bile ducts.

**Pathogenesis**

I. Metabolic:

Cholesterol is synthesised in liver. Its solubility is determined by relative concentration of **cholesterol, bile salts** and **lecithin**. Altered levels of cholesterol, lecithin, and bile salts in bile reduces the micelle concentration in the bile leading to precipitation of insoluble cholesterol, hence, the stone formation (Lithogenic bile).

- Normal ratio of bile salt and lecithin to cholesterol is 25:1. Ratio below 13:1 leads to precipitation of cholesterol. Insoluble cholesterol is within the soluble micelle which is formed by lecithin and bile salts. If cholesterol component increases bile gets supersaturated and inadequate micelle makes insoluble cholesterol to undergo crystallisation and cholesterol monohydrate stone formation

(Admiron's triangular hypothesis).

- Some cholesterol remains as bilayered lipid vesicles which are soluble. A specific heat labile glycoprotein in bile induces cholesterol monohydrate crystal formation in the vesicle and causes their aggregation. It is called as nucleation.

- Eventual precipitation and stone formation occurs by infection/infestation; pancreatic fluid reflux into CBD causing conversion of toxic lecithin to lysolecithin which is also toxic (causes supersaturated bile); bile stasis or altered enterohepatic circulation.

- Any condition which either increases the cholesterol secretion in the bile or reduces the bile salt concentration causes cholesterol stone formation. Old age; OCP; obesity; clofibrate may increase cholesterol secretion. Oestrogen, ileal resection and cholestyramine reduce the bile salt concentration.

- Chenodeoxycholic acid and ursodeoxycholic acid prevent cholesterol stone formation by maintaining bile acid pool; reducing cholesterol synthesis and secretion; converting supersaturated bile into normal bile.

## II. ***Infections and Infestations:***

Bacteria like *E. coli, Salmonella,*

Parasites like *Clonarchis sinensis* and *Ascaris lumbricoides*

are often associated in the formation of Gallstones

## III. ***Bile stasis:***

Occurs due to estrogen therapy, pregnancy,vagotomy and in patients who are on long term intravenous fluids or TPN.

## IV. ***Increased bilirubin production*** due to any of the causes

of haemolysis as in hereditary spherocytosis, sickle cell anaemia, thalassaemia, malaria, cirrhosis. Here pigment stones are common.

## Effects of the Gallstones:

### *In the gallbladder*

a. Silent asymptomatic stones occurs in 10% of males and 20% of females.

b. *Biliary colic with periodicity*, severe within hours after meal (commonest presentation). Biliary colic is spasmodic pain often severe, in right upper quadrant and epigastrium radiating to chest, upper back and shoulder. It is self-limiting, recurs unpredictably, often precipitated by a fatty/heavy meal. Fever and increased WBC count may be observed.

iii. Acute cholecystitis.

iv. Chronic cholecystitis.

v. Empyema gallbladder.

vi. Perforation causing biliary peritonitis or pericholecystitic abscess.

vii. Mucocele of gallbladder.

viii. Limey gallbladder.

ix. Carcinoma gallbladder.

***In the CBD***

- Secondary CBD stones (occurs in 10% of gallstones).
- Acute Cholangitis.
- Acute Pancreatitis.
- *Mirizzi syndrome* (compression of CBD by stone from cystic duct or cholecysto-choledochal fistula).

**In the intestine:**

Cholecystoduodenal fistula causing *gallstone ileus* and so intestinal obstruction,by obstruction of small bowel at the ileo-caecal junction.

**Diagnosis of Gallstone Disease:**

Presence of gallstone is diagnosed by **ultrasound scanning** and **cholangiography.** Cholangiography is the radiological study of biliary ducts after the administration of a contrast medium.

It is now more common to use **Contrast enhanced CT scan** of the Abdomen and pelvis to visualise the Gallstones and CBD stones.

The Next Recent Modality of use is

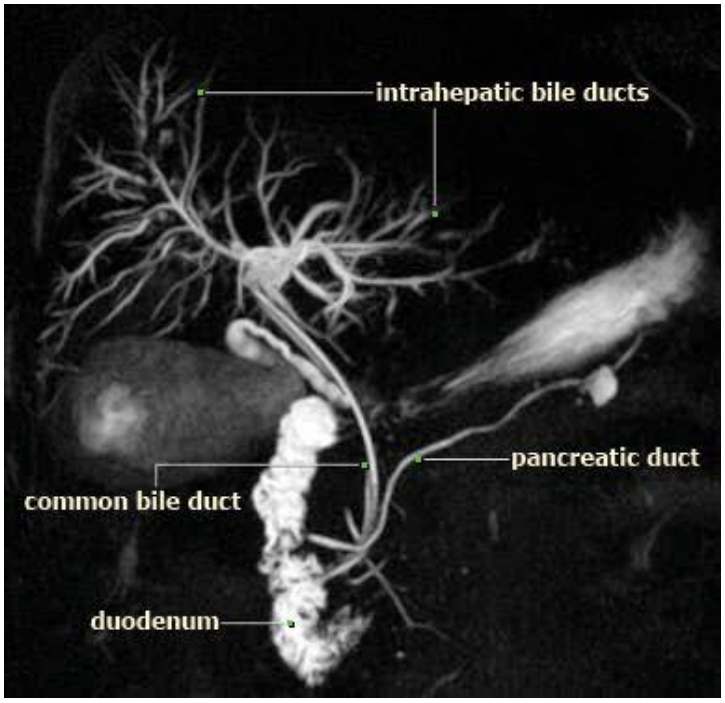
**Magnetic Resonance Cholangio Pancreatography(MRCP).**

It is an excellent modality to map the pathology in the biliary tree

Whether it be gallstones or carcinoma or Stricture involving the

Biliary tree. It has the advantage of non radiating and non invasive

Modality.



**Endoscopic Retrograde Pancreatico Cholangiography(ERCP)** is

Vital diagnostic as well as therapeutic tool in evaluating and treating

Distal biliary tree impacted stones or growth.

It has an added advantage of being therapeutic

also. Endoscopy guided removal of stone and sphincterotomy can

be done in the same sitting as a single procedure.

## INFECTIONS OF THE BILIARY TREE :

## ACUTE CHOLECYSTITIS :

Acute Cholecystitis is an acute abdominal emergency that is usually a result of gallbladder outlet being obstructed due to some etiology leading to bile stasis and infection of the retained bile & clinically patient presenting with Fever, upper abdominal pain and a palpable tender Gall Bladder**.**

## Classification:

1. Acute calculous cholecystitis.
2. Acute acalculous cholecystitis

## Pathogenesis of Acute Cholecystitis:

Stone causes obstruction at Hartmann's pouch or in cystic duct. Obstruction causes stasis, oedema of the wall, bacterial infection, acute cholecystitis and its effects. Impacted stone also causes mucosal erosion allowing bile salts to act over the submucosal tissues as bile is toxic to these tissues. It leads into necrosis, further infection and often perforation of the gallbladder usually at Hartmann's pouch.

## Clinical Features

Sudden onset of pain in the right hypochondrium, with

tenderness, guarding, and rigidity.

Palpable, tender, smooth, soft gallbladder.

Area of hyperaesthesia between 9th and 11th ribs posteriorly

on the right side (*Boas's sign)*.

Jaundice may/ may not be present**.**

Fever, nausea, palpable tender mass in GB region (25%).

Tachycardia and toxic features.

MURPHY'S SIGN: It is a characteristic clinical sign that can be elicited in a patient with inflamed Gall Bladder.With a palpating hand over the gallbladder point in the Right Hypochondrium when the patient is asked to take a deep breath,there is a characteristic inspiratory catch which is classic Murphy's sign.

The same can be elicited in the patient while doing an Ultrasound

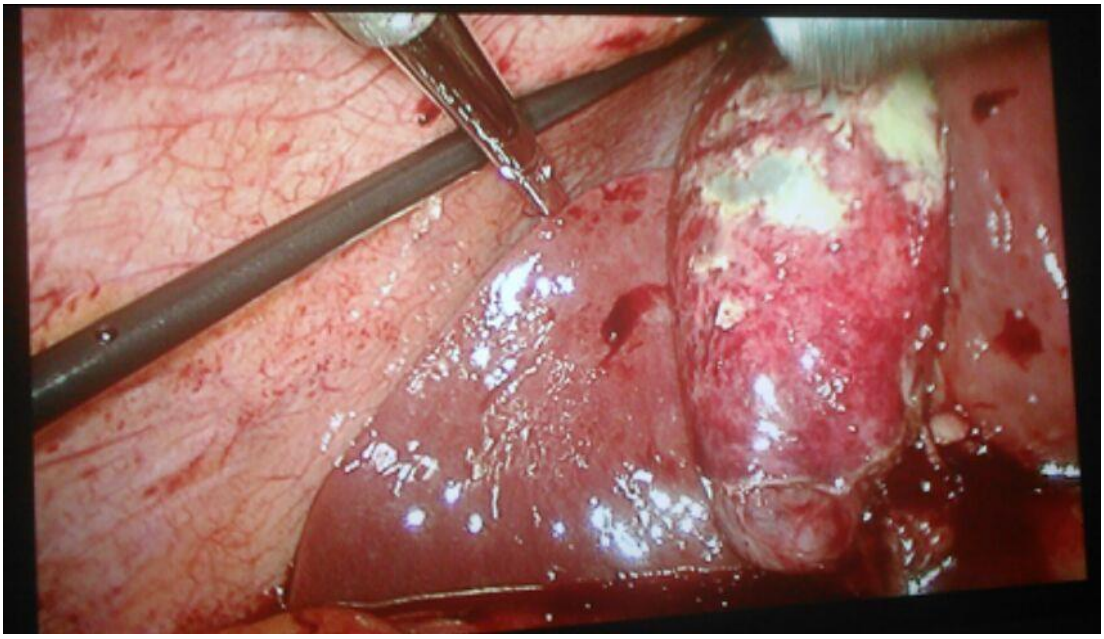
This is called as ULTRASONOGRAPHIC MURPHY'S SIGN.

## VARIANTS OF ACUTE CHOLECYSTITIS:

Acute Cholecystitis can present clinically as variant forms which are distinct in their own type of presentation and specific age group and
Severity of the disease and significantly decide the outcomes .

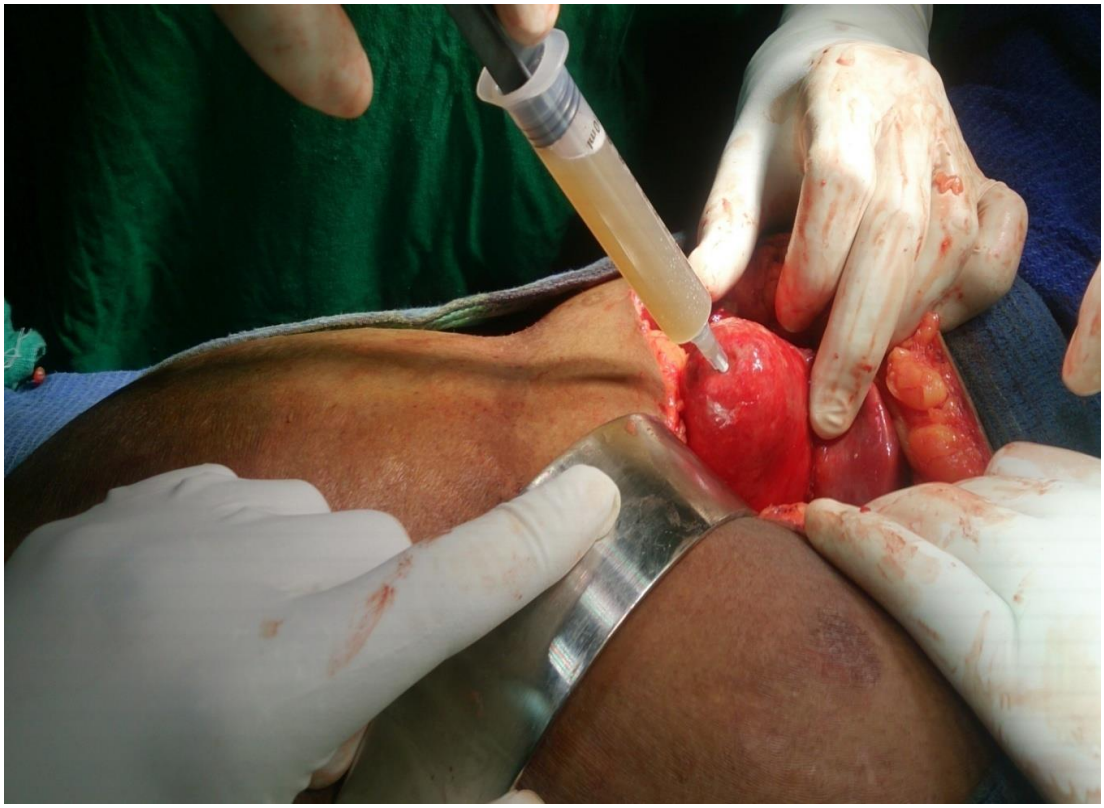
## Variants include:

- ✓ Acute Emphysematous Cholecystitis which presents in older age group and more commoner among diabetics and immunocompromised .It is caused by gas forming organisms in pathogenesis namely Clostridia.This clinical variant needs early recognition and early surgical treatment as delay in diagnosis and treatment leads to complications and significant increase in mortality and morbidity.



- ✓ Acute Gangrenous Cholecystitis:

This clinical variant of acute cholecystitis is characterized by the presence of early necrosis of gallbladder wall and early perforation of gall bladder resulting peritonitis and sepsis.This also needs prompt recognition and early treatment.



- ✓ Empyema Gallbladder:

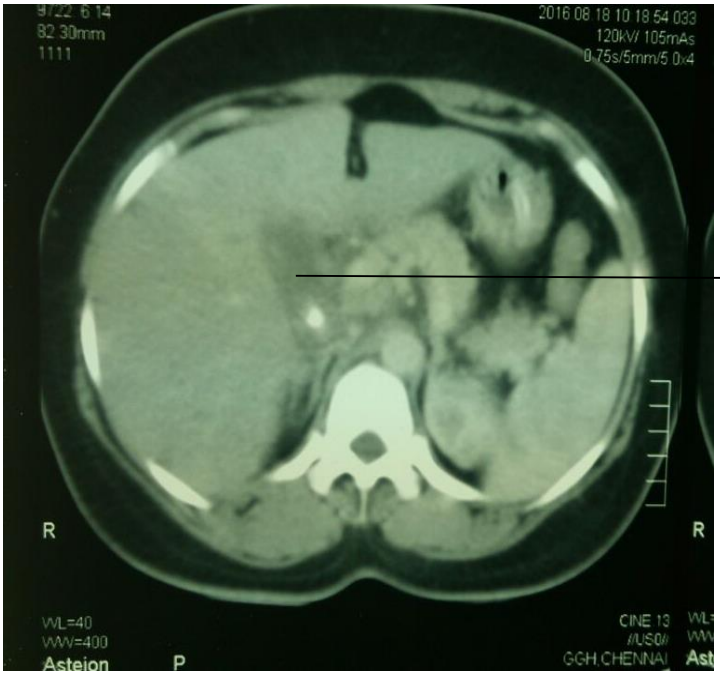
This variant of acute cholecystitis is more aggressive variety and is charcterised by distension of gallbladder with acute inflammatory pus as a result of more aggressive course of infections.Empyema Gallbladder demands early surgical treatment as this variant is unlikely to respond to conventional antibiotic therapy.

Differential diagnosis to be considered while suspecting ACUTE CHOLECYSTITIS.

- Duodenal ulcer perforation.
- Acute pancreatitis.
- Acute appendicitis.
- Acute pyelonephritis.
- Acute Hepatitis.
- Lobar pneumonia, myocardial infarction.

## DIAGNOSIS:

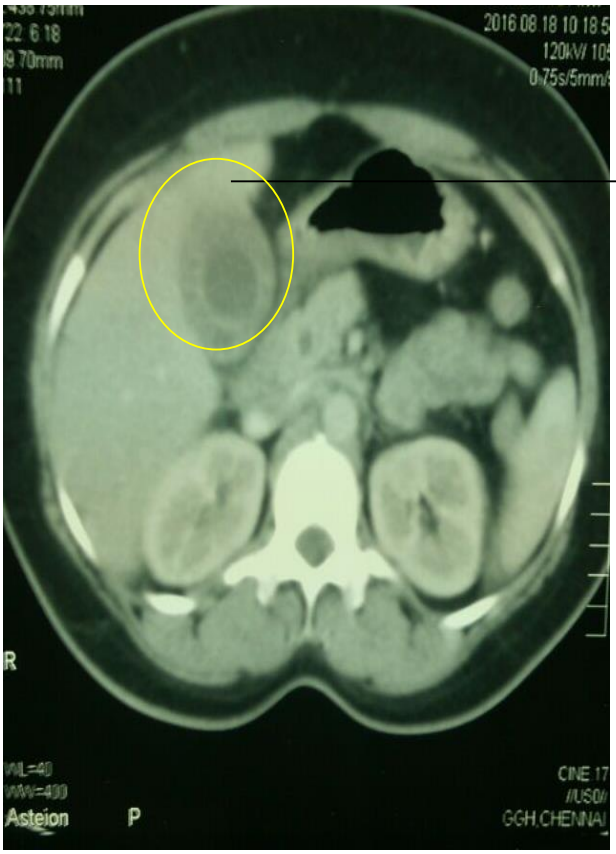
Acute Cholecystitis is an acute surgical emergency.Diagnosis made with Help of clinical ,imaging and lag findings after exclusion of other possible differentials as discussed above.




CT PICTURE SHOWING DISTENDED GALLBLADDER AND VISUALISATION OF RADIOOPAQUE GALLSTONE AT THE NECK

## MANAGEMENT:

Acute Cholecystitis is Managegd by Surgery which can be either early or can be Delayed after the initial inflammatory surge has settled.Appropriate sensitive IV antibiotics are instituted targeting the most common flora of the biliary tract namely Gram negative enterococci such as Eschierichia Coli.,Klebsiella species ,Proteus and others. Early surgical treatment can be accomplished by Laparascopic cholecystectomy or Conventional open cholecystectomy. Sometimes a even conservative surgical technique such as subtotal cholecystectomy may also be done.




DISTENDED GALLBLABBER WITH GB WALL THICKENING IN CT SCAN.

## ACUTE CHOLANGITIS:

Acute bacterial cholangitis is a serious life-threatening emergency caused by infection of an obstructed biliary tract. The systemic manifestations result from bacteraemia.

The most common obstructing agent is an occluding

stone in the common bile duct, followed by bile duct strictures (including sclerosing cholangitis) and, less commonly, tumours of the bile ducts, pancreatic head and periampullary lesions. Cholangitis may also complicate bilioenteric anastomoses, spontaneous bilioenteric fistulae, cystic disease of the biliary tract and duodenal diverticulae. Cholangitis may also occur following instrumentation of the biliary tract, e.g. after ERCP. The infection is most commonly caused by Gram negative

organisms.

The classic triad of symptoms consists of

- ✓ pain in the right hypochondrium,
- ✓ intermittent fever and
- ✓ jaundice

(Charcot's Triad).

Aside from toxicity, the high intermittent pyrexia is accompanied by severe rigors. The pain varies in intensity and may be severe. There is usually

tenderness in the right hypochondrium that, if marked, suggests the presence of abscess formation. Nausea and vomiting are frequent accompaniments.

Hypotension is found in patients with severe cholangitis, when renal failure is usually present. The overall reported mortality of patients requiring urgent decompression for severe cholangitis is 15-20%.

## DIAGNOSIS:

High index of clinical suspicion and imaging evidence of dilated biliary tree and stagnant bile is more in favour of Acute cholangitis.Lab parameters in the form of increased serum bilirubin And Serum Alkaline phosphatase and total leucocyte count are all more in favour the Diagnosis.

## MANAGEMENT:

In acute cholangitis in addition to clinical sepsis most of the patients present with multiple organ dysfunction and are in a state of shock. Treatment of Acute cholangitis is to be more focused on providing support for organ dysfunction in the form of intensive supportive care and definitive procedure to decompress the biliary tree is planned henceforth .

Decompression of the obstructed and dilated biliary tract is done

By either surgically or percutaneous or endoscopic guided depending upon the status of the patient and the level of obstruction in the biliary tree.

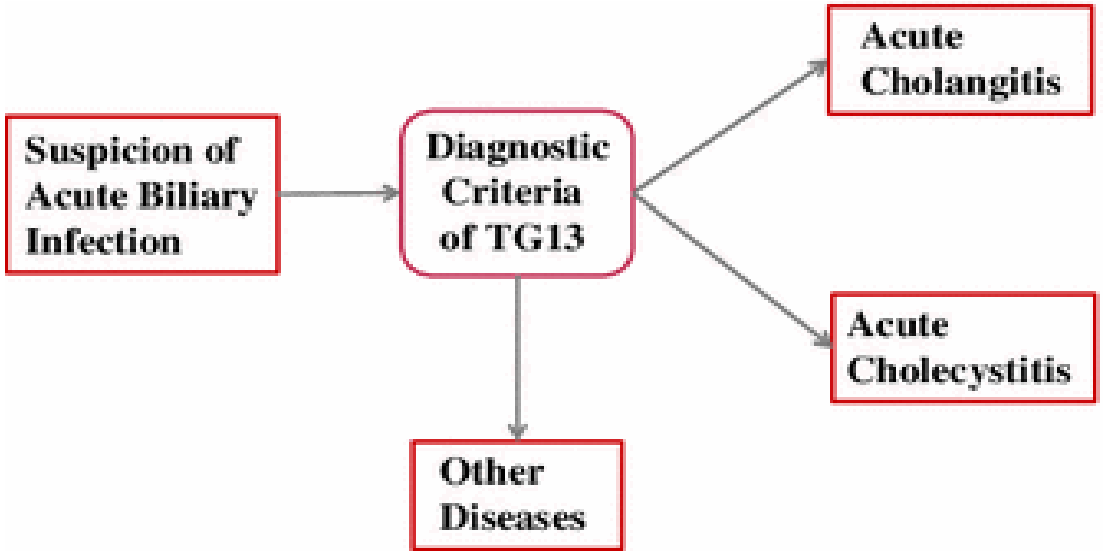
Appropriate IV antibiotics to be instituted depending the flora and septicaemia status of the patient.Organ dysfunction should be promptly addressed in a intensive care setting and close monitoring of vitals done.

## TOKYO GUIDELINES 2013 IN MANAGEMENT OF ACUTE BILIARY INFECTIONS:

In 2003,The Japanese society of hepato-pancreatic biliary surgery formed working group to formulate guidelines to diagnose and manage the acute biliary sepsis.As a result of which Tokyo guidelines 2007 was formulated dictating diagnostic criteria and severity assessment criteria for protocol based management of acute cholecystitis and acute cholangitis.There were few flaws and drawbacks in 2007 format,hence it was reworked upon and updated Tokyo guidelines was formulated in the year 2013.

This Tokyo guidelines 2013 is the currently available only guidelines universally in the management of above said biliary infections.

The guidelines not only aids in the diagnosis of Acute cholecystitis and Acute cholangitis but also offers a platform to assess the severity of the disease with which the patient presents to medical attention and guide appropriate treatment.


Suspicion of Acute Biliary Infection
Diagnostic Criteria of TG13
Acute Cholangitis
Acute Cholecystitis
Other Diseases

# TG 13 DIAGNOSTIC CRITERIA FOR ACUTE CHOLECYSTITIS

| | | |
|---|---|---|
| **A** | **LOCAL SIGNS OF INFLAMMATION:** | |
| **A-1** | Murphy's sign | ☐ |
| **A-2** | Right upper quadrant pain/mass/tenderness | ☐ |
| **B** | **SYSTEMIC SIGNS OF INFLAMMATION:** | |
| **B-1** | Fever | ☐ |
| **B-2** | Elevated CRP | ☐ |
| **B-3** | Elevated WBC counts | ☐ |
| **C** | **IMAGING:** | |
| **C-1** | Thickening of Gallbladder > 5mm | ☐ |
| **C-2** | Enlarged Gallbladder | ☐ |
| **C-3** | Debris Echo | ☐ |
| **C-4** | Ultrasonographic's Murphy's Sign | ☐ |
| **C-5** | Gas imaging | ☐ |
| **C-6** | Pericholecystic fluid | ☐ |

**SUSPECTED DIAGNOSIS: One item in A + One item in B** ☐

**DEFINITE DIAGNOSIS : One item in A + One item in B +C** ☐

## SEVERITY ASSESSMENT CRITERIA FOR ACUTE CHOLECYSTITIS:

### GRADE III : ACUTE CHOLECYSTITIS + Any of following organ dysfunction :

| | | |
|---|---|---|
| **1.** | **CVS Dysfunction:**<br>**Hypotension requiring treatment with dopamine>/= 5µg/kg/min or any dose of Noradrenaline** | ☐ |
| **2** | **Neurological Dysfunction:**<br>**Decreased level of consciousness** | ☐ |
| **3.** | **Renal Dysfunction:**<br>**Oliguria/ creatinine > 2 mg/dl** | ☐ |
| **4** | **Respiratory Dysfunction:**<br>**PaO2/FiO2 ratio < 300** | ☐ |
| **5** | **Hepatic dysfunction**<br>**PT-INR > 1.5** | ☐ |
| **6.** | **Hematological Dysfunction:**<br>**Platelet count < 1,00,000/mm 3** | ☐ |

### Grade II: Presence of any of the following:

| | | |
|---|---|---|
| **1** | **Elevated WBC count > 18,000/mm3** | ☐ |
| **2** | **Palpable tender mass in Right upper Quadrant** | ☐ |
| **3** | **Duration of symptoms > 72 hours** | ☐ |
| **4** | **Marked local inflammation(gangrenous or emphysematous cholecystitis/pericholecystic or hepatic abscess/biliary peritonitis)** | ☐ |

### GRADE I: Does not meet the criteria of GRADE III/GRADE II. ☐

## TG 13 CRITERIA FOR DIAGNOSING ACUTE CHOLANGITIS

| | | |
|---|---|---|
| **A** | **SYSTEMIC INFLAMMATION:** | |
| **A-1** | Fever and /or shaking chills | ☐ |
| **A-2** | WBC Count < 4000 / >10000 cells/mm3 or<br>CRP .>/= 1mg/dl | ☐ |
| **B** | **CHOLESTASIS** | |
| **B-1** | Jaundice (T.Bilirubin > 2mg/dl) | ☐ |
| **B-2** | Abnormal Liver function tests | ☐ |
| **C** | **IMAGING:** | |
| **C-1** | Biliary dilatation | ☐ |
| **C-2** | Evidence of etiology on imaging<br>(stricture/stone/stent) | ☐ |

**SUSPECTED DIAGNOSIS : One item in A + One item in B** ☐

**DEFINITE DIAGNOSIS :One item in A + One item in B + One Item in C** ☐

## SEVERITY ASSESSMENT CRITERIA FOR ACUTE CHOLANGITIS:

### GRADE III : ACUTE CHOLANGITIS + Any of following organ dysfunction :

| | | |
|---|---|---|
| 1 | **CVS Dysfunction:**<br>Hypotension requiring treatment with dopamine>/= 5µg/kg/min or<br>any dose of Noradrenaline | ☐ |
| 2 | **Neurological Dysfunction:**<br>Decreased level of consciousness | ☐ |
| 3 | **Renal Dysfunction:**<br>Oliguria/ creatinine > 2 mg/dl | ☐ |
| 4 | **Respiratory Dysfunction:**<br>PaO2/FiO2 ratio < 300 | ☐ |
| 5 | **Hepatic dysfunction**<br>PT-INR > 1.5 | ☐ |
| 6 | **Hematological Dysfunction:**<br>Platelet count < 1,00,000/mm 3 | ☐ |

### Grade II: Presence of any of the following:

| | | |
|---|---|---|
| 1 | Elevated WBC count > 12,000/mm3 & < 4000 cells /mm3 | ☐ |
| 2 | High fever (>39 degree centigrade) | ☐ |
| 3 | Age > / = 75 years | ☐ |
| 4 | Hyperbilirubinemia (T.bilirubin >5 mg/dl) | ☐ |
| 5 | Hypoalbuminemia (< Max upper limit value x 0.7) | ☐ |

### GRADE I: Does not meet the criteria of GRADE III/GRADE II at Initial diagnosis ☐

**MANAGEMENT OF CHOLECYSTITIS ACCORDING TO TG 13 GUIDELINES**:

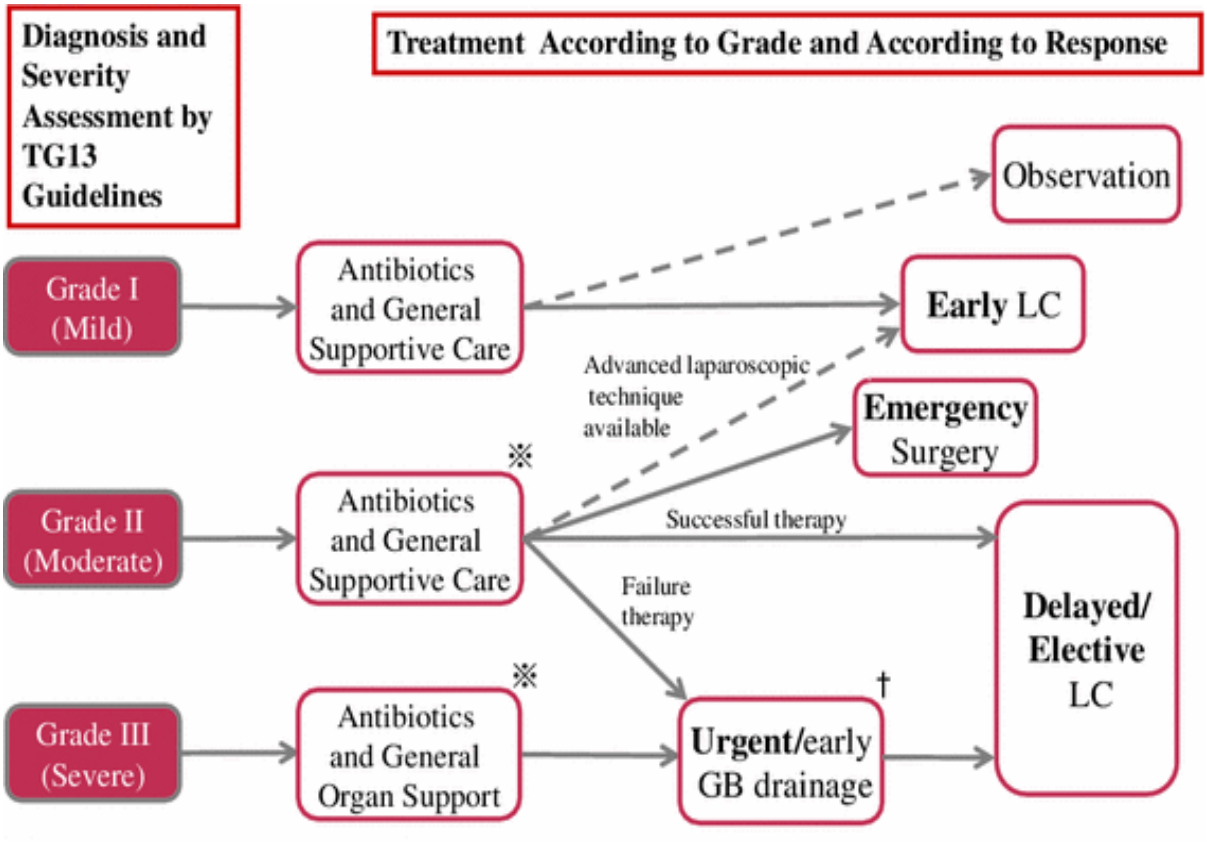
**Grade I (Mild) acute cholecystitis**: Early laparoscopic cholecystectomy is the preferred procedure.

**Grade II (Moderate) acute cholecystitis**: Early cholecystectomy is recommended in experienced centers. However, if patients have severe local inflammation, early gallbladder drainage (percutaneous or surgical) is indicated. Because early cholecystectomy may be difficult, medical treatment and delayed cholecystectomy are necessary.

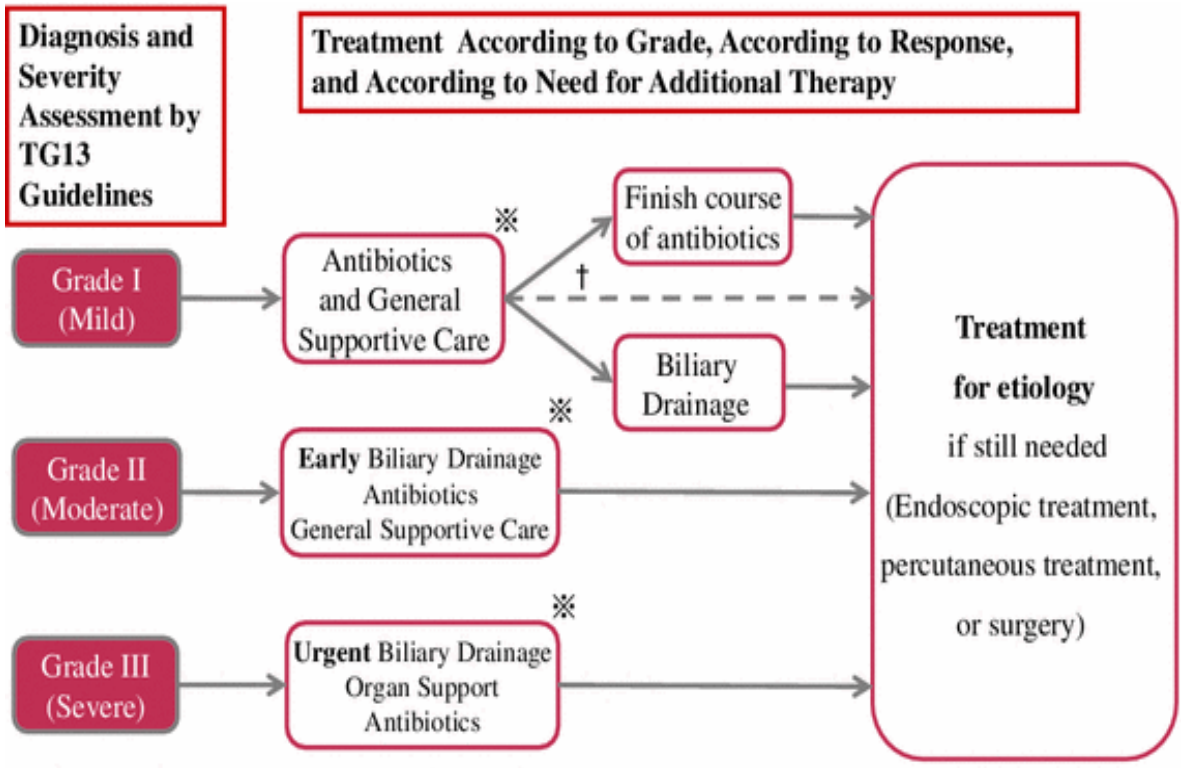
**Grade III (Severe) acute cholecystitis**:

Urgent management of organ dysfunction and management of severe local inflammation by gallbladder drainage should be carried out. Delayed elective cholecystectomy should be performed when cholecystectomy is indicated.

GUIDELINE STRATEGY FOR MANAGING ACUTE CHOLECYSTITIS DEPENDING UPON THE GRADE OF THE DISEASE:



GUIDELINE STRATEGY FOR MANAGING ACUTE CHOLANGITIS DEPENDING UPON THE GRADE OF THE DISEASE:



# RESULTS

LIST OF ANALYSIS IN ACUTE CHOLECYSTITIS:

TOTAL NUMBER OF PATIENTS ANALYSED: 23

1.Age wise incidence.

2.Sex Distribution.

3.Duration of Symptoms in the study group.

4.Symptoms in the study group.

5.Signs in the study group.

6.Ultrasonogram findings in the study group.

7.Biochemical Results in the study group.

8.Incidence of Diabetes among study population.

9. Etiology among study group.

10.Pattern of Gallstone disease in study group.

11.Analysis of the Grade of disease among the study group.

12.Age vs Grade analysis in study group.

13.Age vs sex correlation among study group.

14.Duration of Diabetes vs Grade of disease.

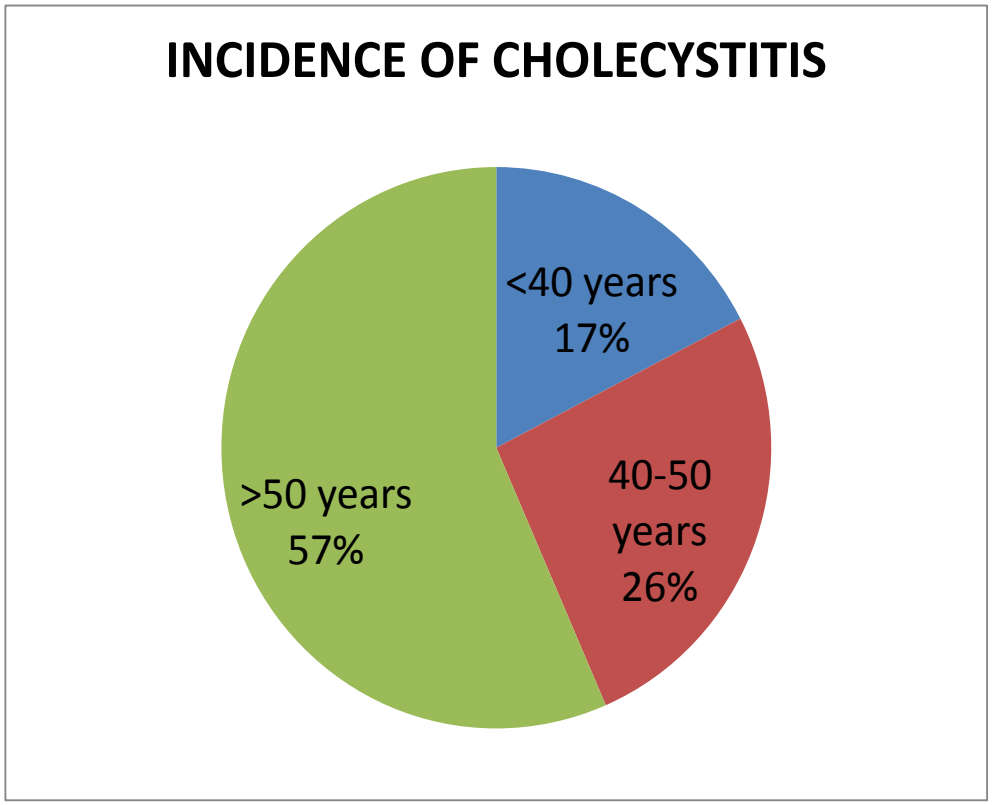
15.Age-grade-incidence of diabetes among study group.

16.Analysis of management outcomes in Grade 1 cholecystitis.

17.Analysis of management outcomes in Grade 2 cholecystitis.

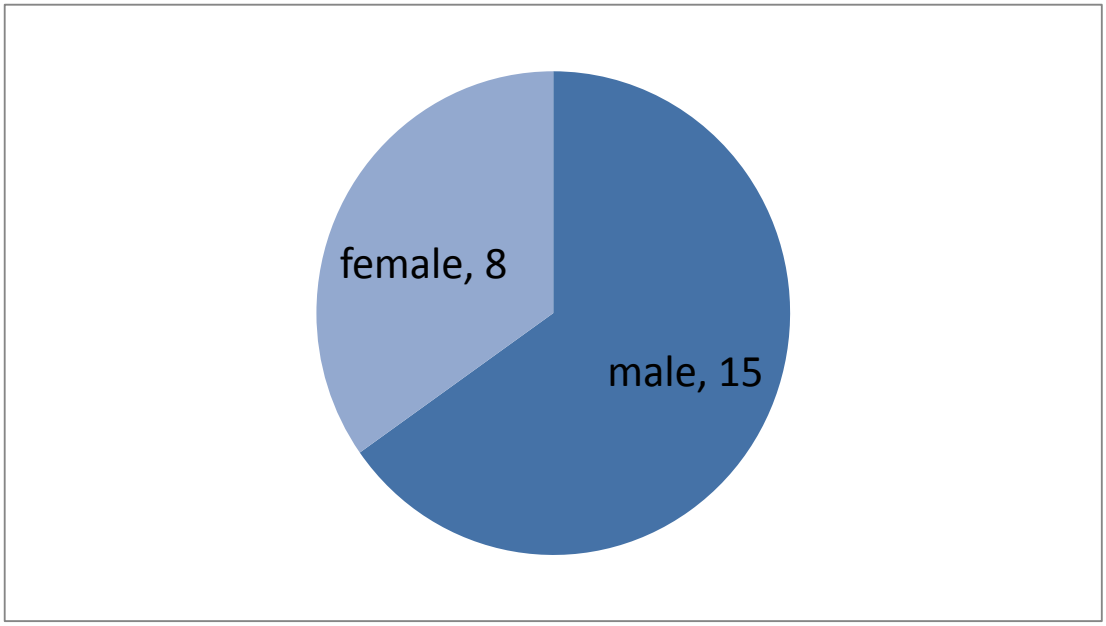
18.Complications in the study group.

AGE WISE INCIDENCE



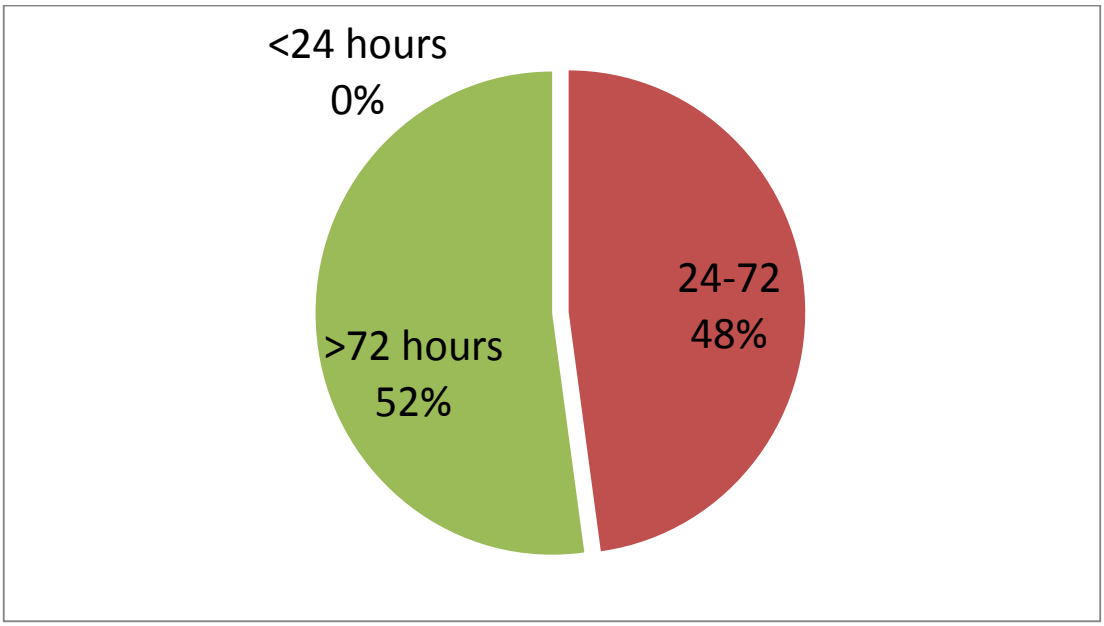

Incidence of acute cholecystitis follows common literature pattern of occurring in elderly patients above 50 years of age.

# SEX DISTRIBUTION




ACUTE CHOLECYSTITIS HAD A HIGHER INCIDENCE IN MALES(65 %) IN OUR STUDY GROUP

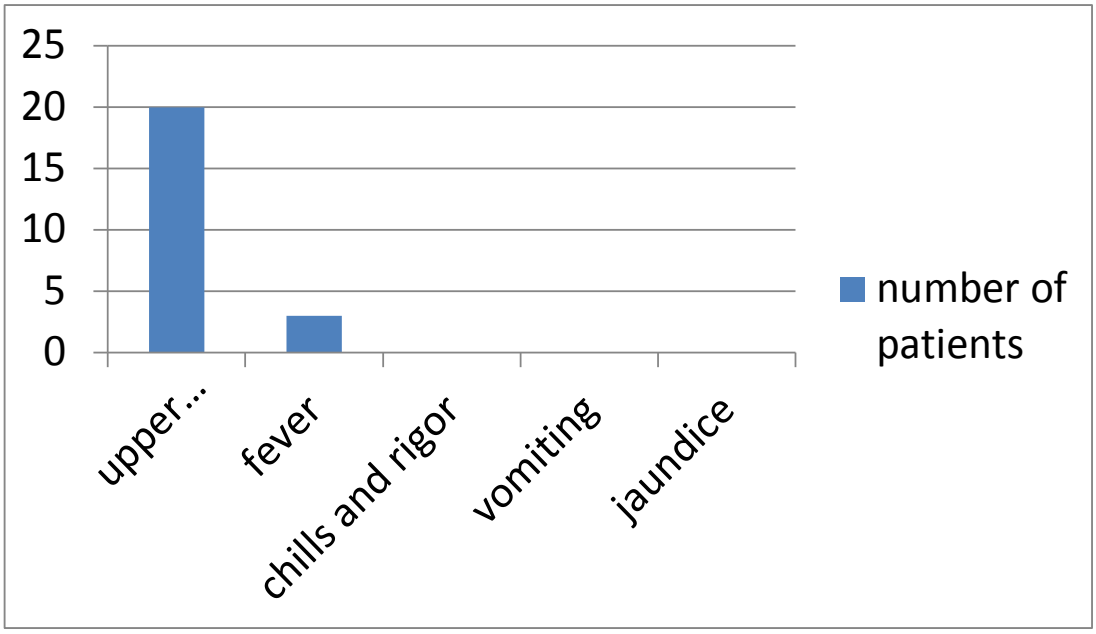
# DURATION OF SYMPTOMS




More than 52 % of the patients presented to medical attention after 72 hours because of lack of awareness and early recognition both by patients and doctors.

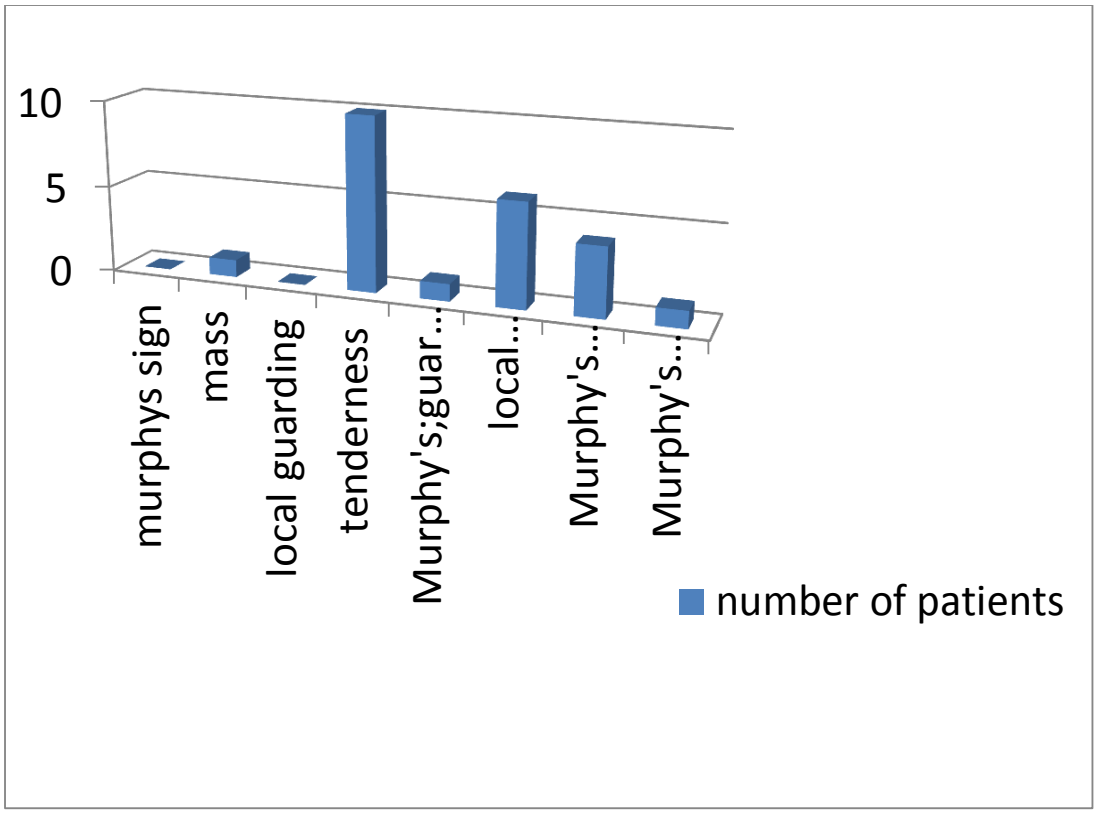
Invariably patient will be categorized as GRADE 2 CHOLECYSTITIS

SYMPTOMS IN STUDY GROUP



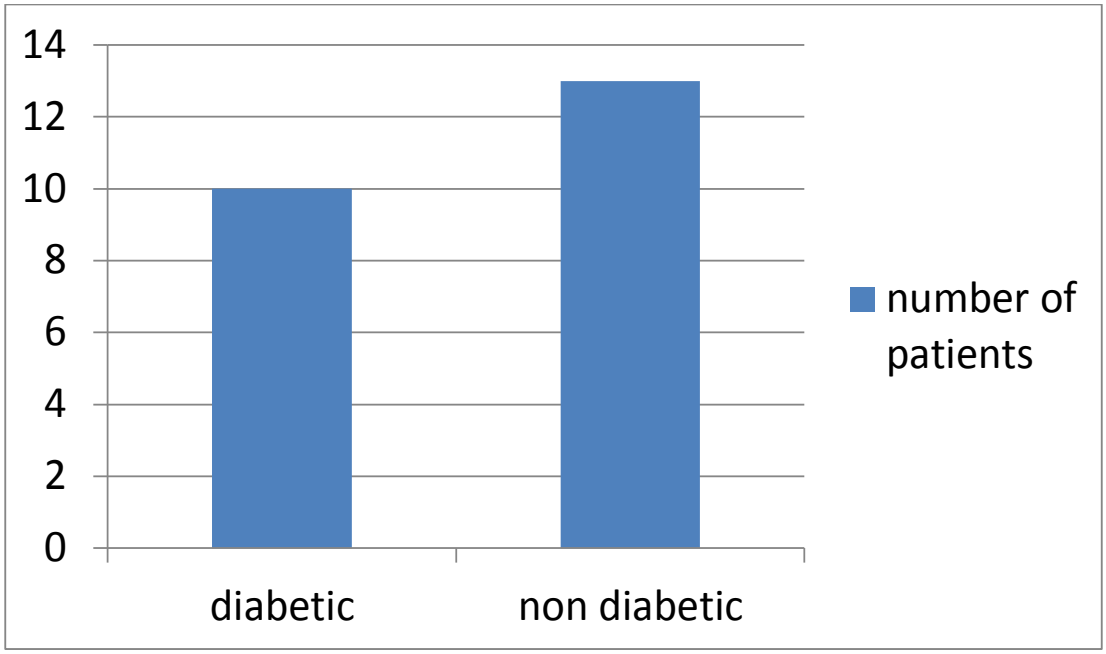

Acute onset Upper abdominal pain more over the Right Hypochondrium is the predominant complaint and was the first to arise in the study population.

CLINICAL SIGNS AMONG THE STUDY POPULATION



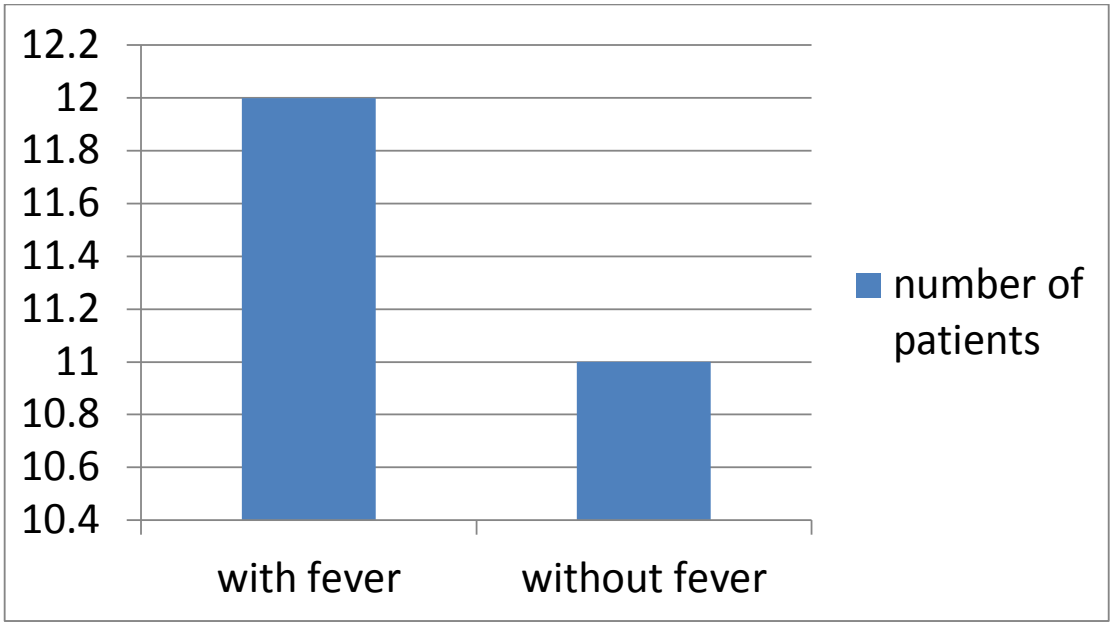

9 out of 13 patients had tenderness in upper abdomen alone as the predominant Clinical sign on examination. Murphy's sign alone was not seen in a single patient.

DIABETIC HISTORY



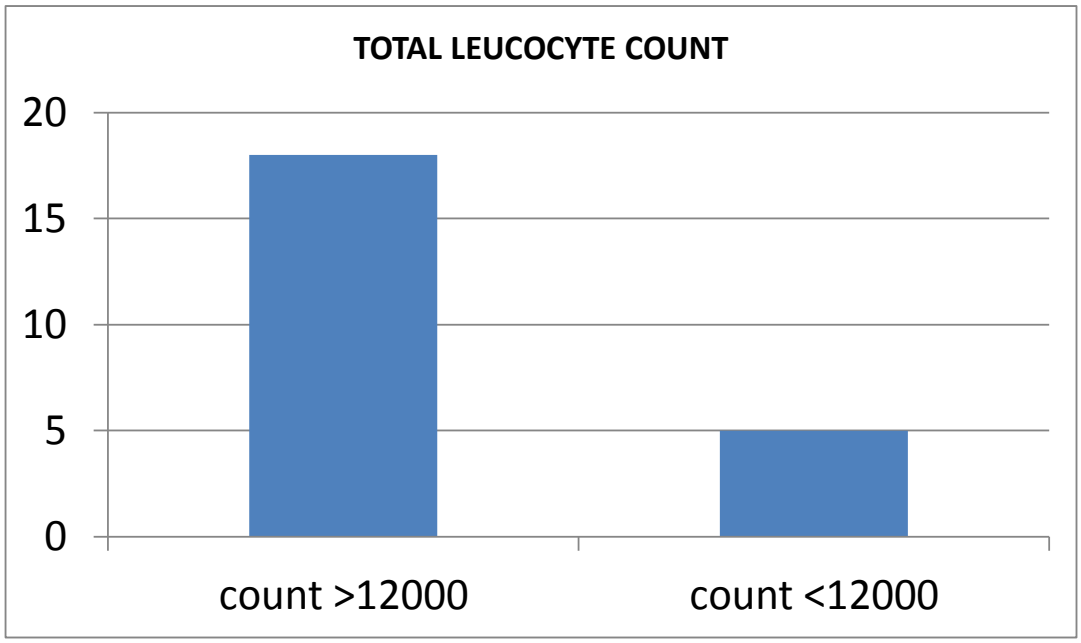

43 % OF THE STUDY POPULATION WERE DIABETIC AND WERE UNDER TRETAMENT FOR THE SAME.

FEVER IN STUDY POPULATION

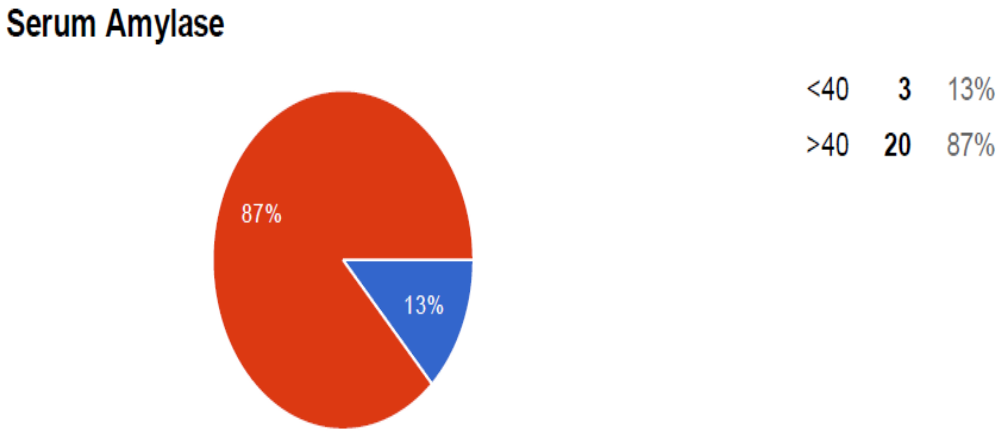



Nearly 50 % of the study group were afebrile at the time of presentation.

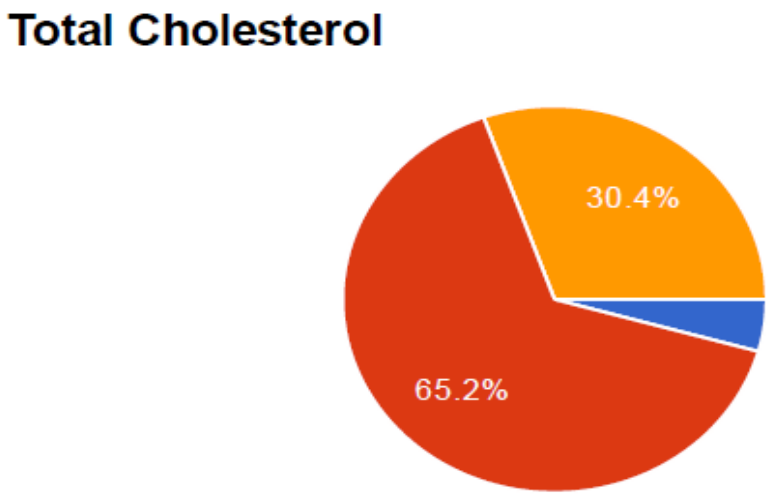
TOTAL LEUCOCYTE COUNT




More than 80 % of the study population had an elevated total Leucocyte count of more than 12000 cells/mm3.



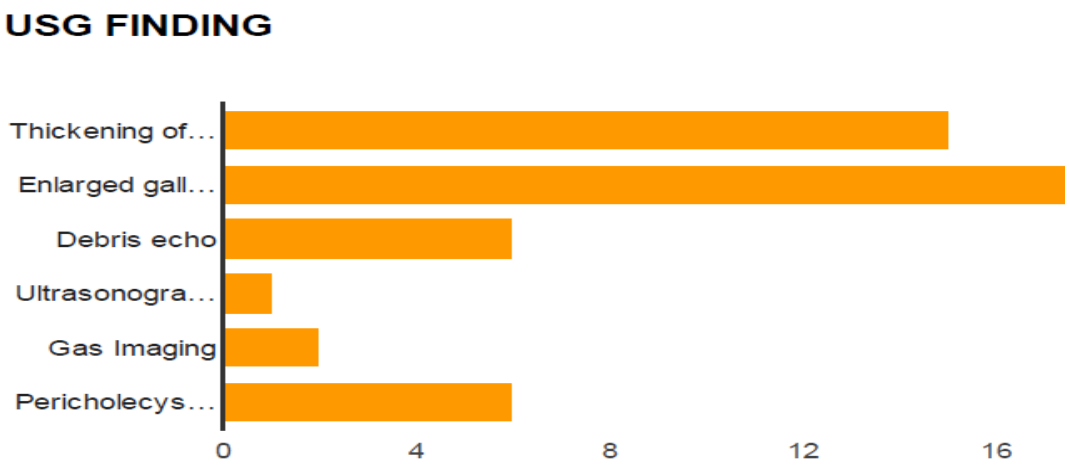

More than 80 % of the study population had serum amylase level Above 40 IU/L.




| | | |
|---|---|---|
| <140 mg/dl | **1** | 4.3% |
| 141-200 mg/dl | **15** | 65.2% |
| >200 mg/dl | **7** | 30.4% |

More than 60 % of the study group had an elevated total Cholesterol level of more than 150 mg/dl.

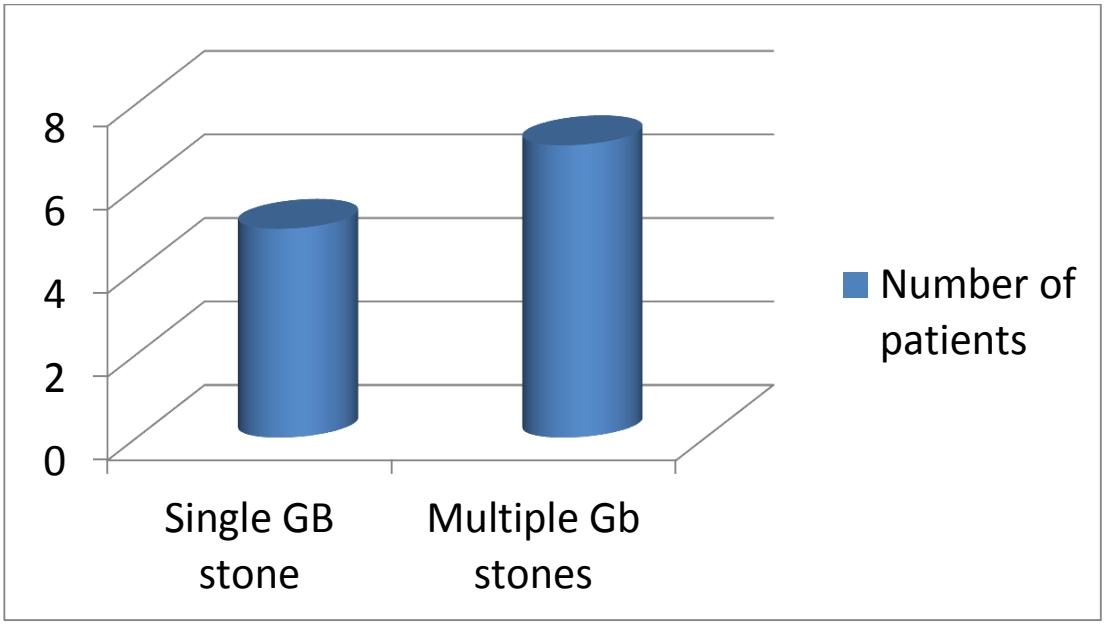
ULTRASONOGRAM FINDING IN STUDY POPULATION




| | | |
|---|---|---|
| Thickening of gallbladder(>5mm) | **15** | 65.2% |
| Enlarged gallbladder | **18** | 78.3% |
| Debris echo | **6** | 26.1% |
| Ultrasonographic's murphy's sign | **1** | 4.3% |
| Gas Imaging | **2** | 8.7% |
| Pericholecystic fluid | **6** | 26.1% |

Thickening of Gall bladder wall (65 %) was the most consistent Finding In ultrasonogram of the study population.Enlarged Gallbladder was the second most common finding that is attributed To Acute cholecystitis.

PATTERN OF GB STONE DISEASE




In most patients with Gallstones as detected etiology Multiple GB stones was more common .In patients with single Gallstone the predominant site was neck of gallbladder.

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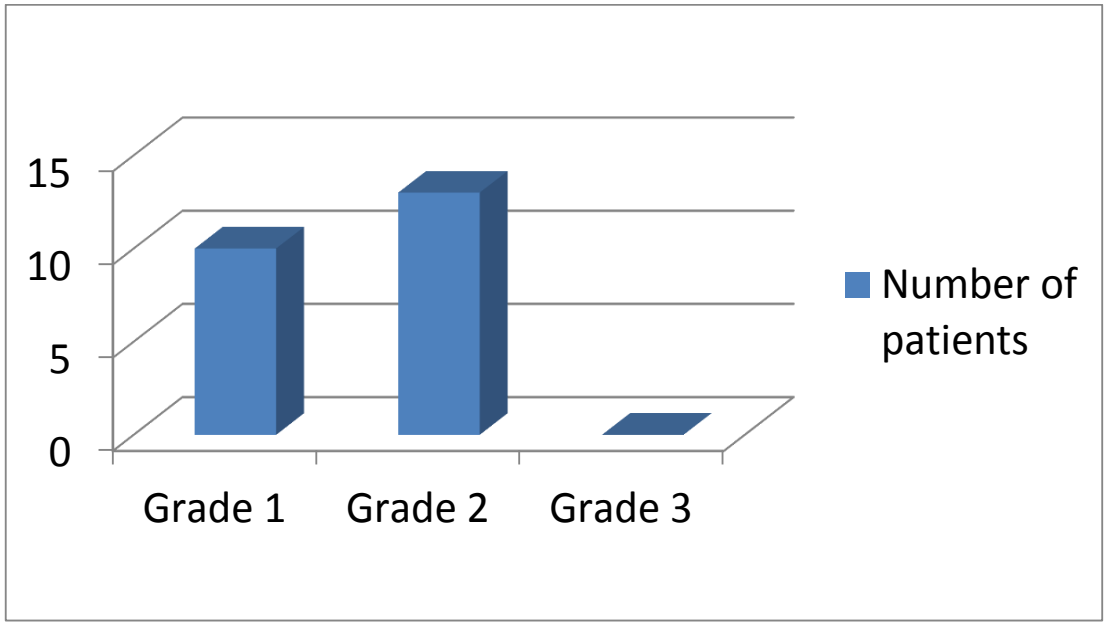
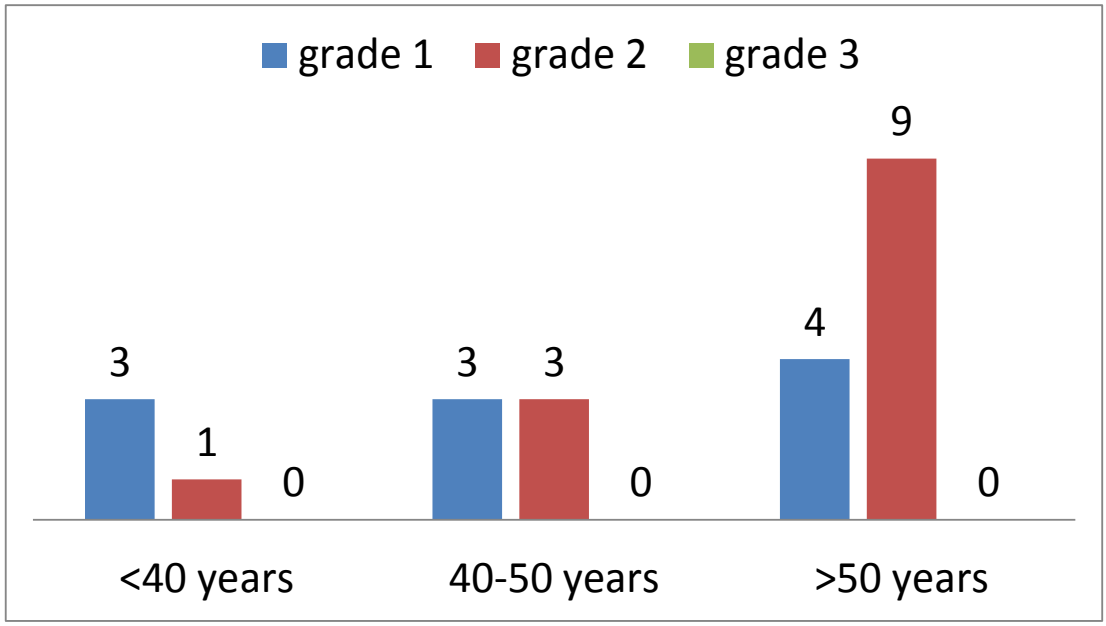
# GRADE OF CHOLECYSTITIS



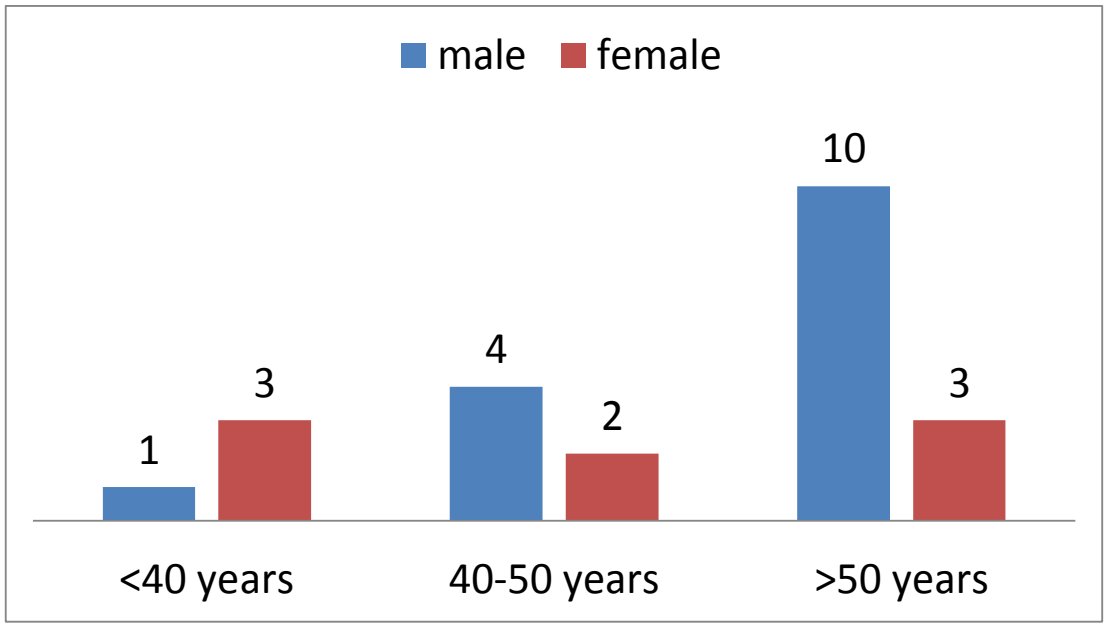

Grade 2 Cholecystitis was the most common Grade of disease of most patients in our study group.More than 50% of study population Had grade 2 disease at the time of presentation.

AGE VS GRADE DISTRIBUTION



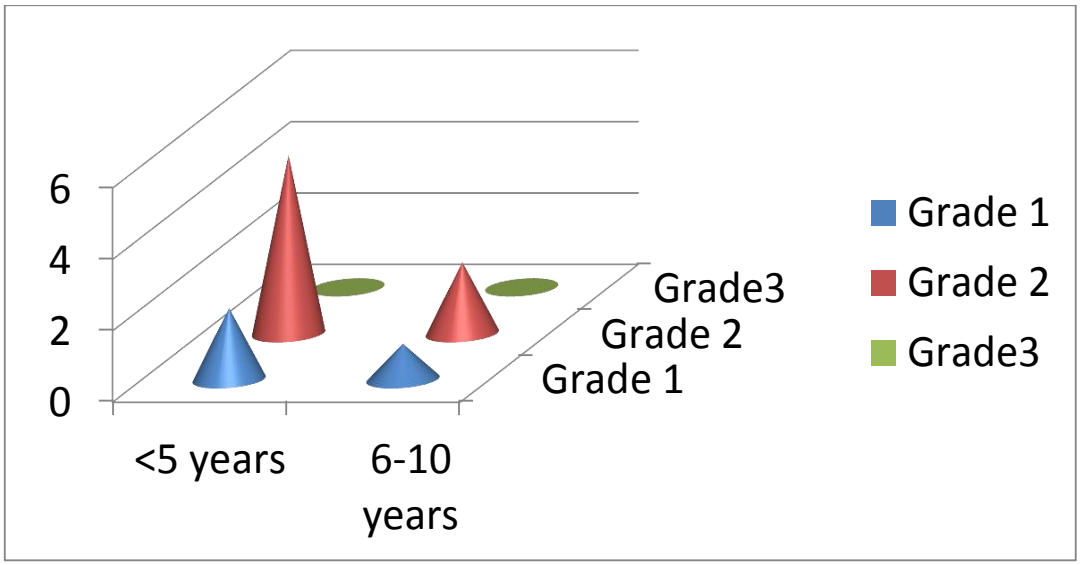

Patients more than 50 years of age have a higher incidence of the disease,with the grade of cholecystitis higher at the time of presentation.

AGE AND SEX CO-RELATION



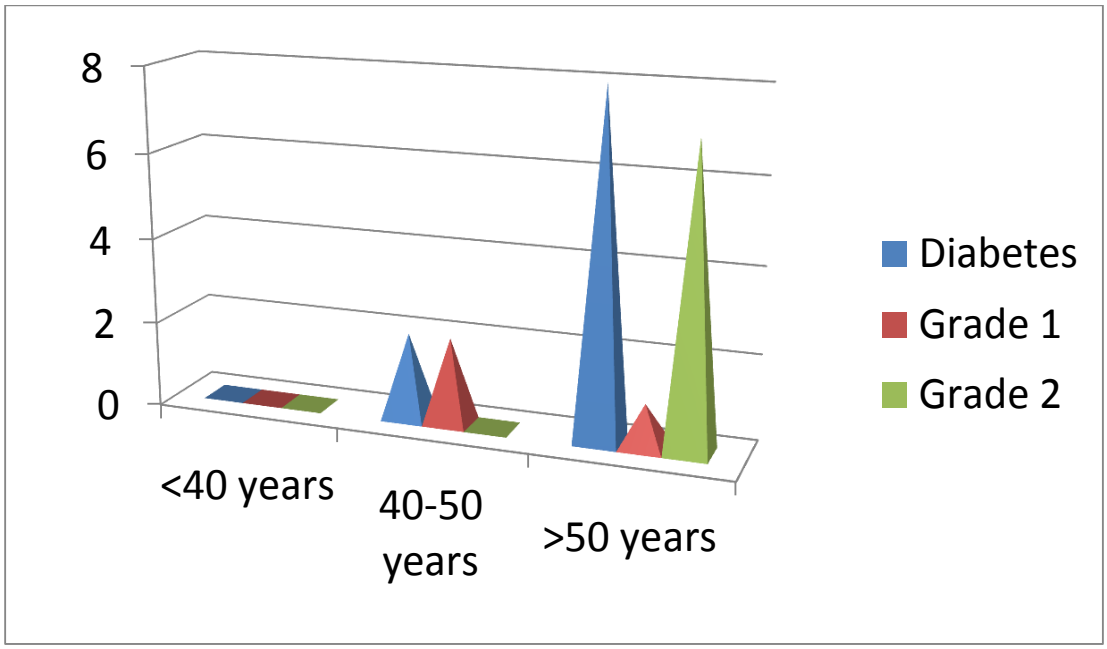

Males more than 50 years of age are higher up in the incidence chart.

DURATION OF DIABETES WITH SEVERITY OF DISEASE




Diabetics of less than 5 years of duration present more with the disease And also a higher grade at presentation.

AGE –INCIDENCE OF DIABETES-GRADE OF DISEASE




Diabetic males above 50 years of age have a higher Incidence of the disease and with a higher grade at the time of presentation.

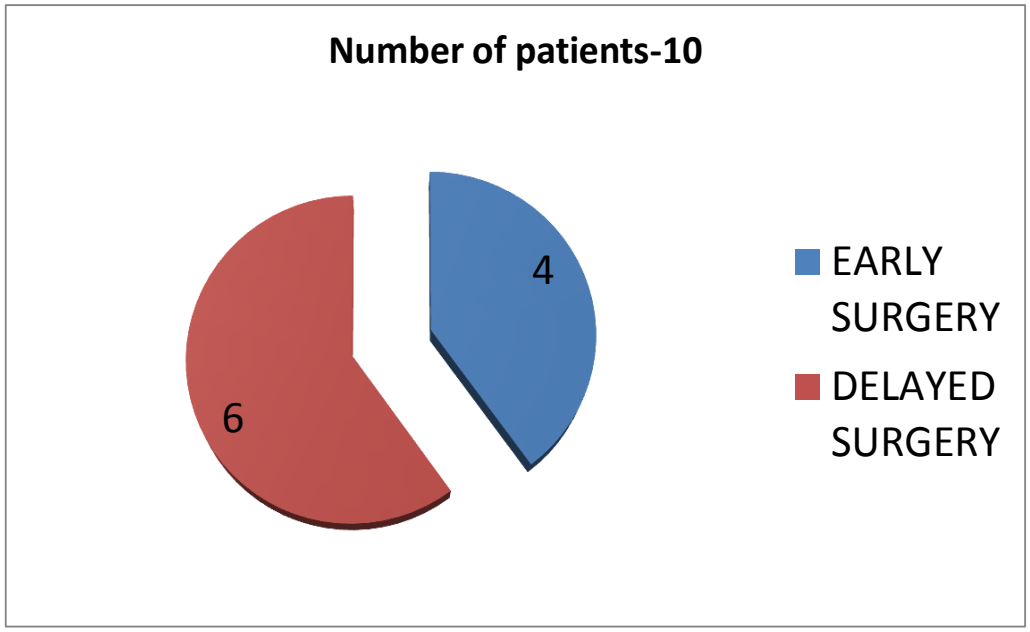
# MANAGEMENT OF GRADE 1

# ACUTE CHOLECYSTITIS

GRADE 1 CHOLECYSTITIS

MANAGEMENT OUTCOMES

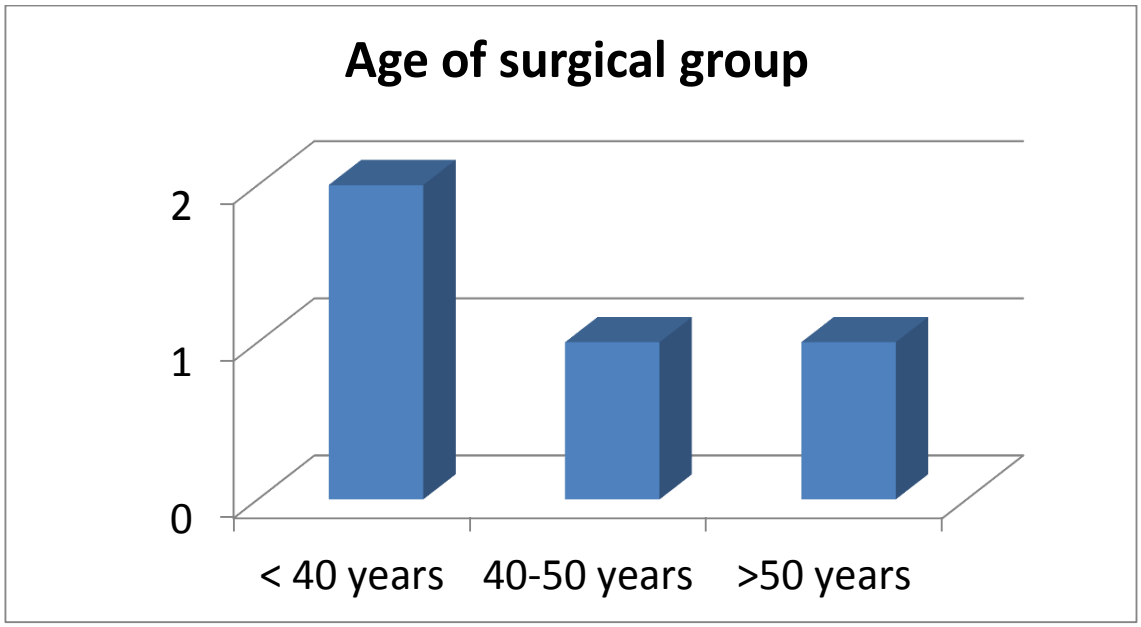
TOTAL NUMBER OF PATIENTS : 10




Most of Grade 1 patients(60 %) were managed Conservatively with antibiotics and delayed surgery .

AGE DISTRIBUTION OF SURGICAL PATIENTS IN

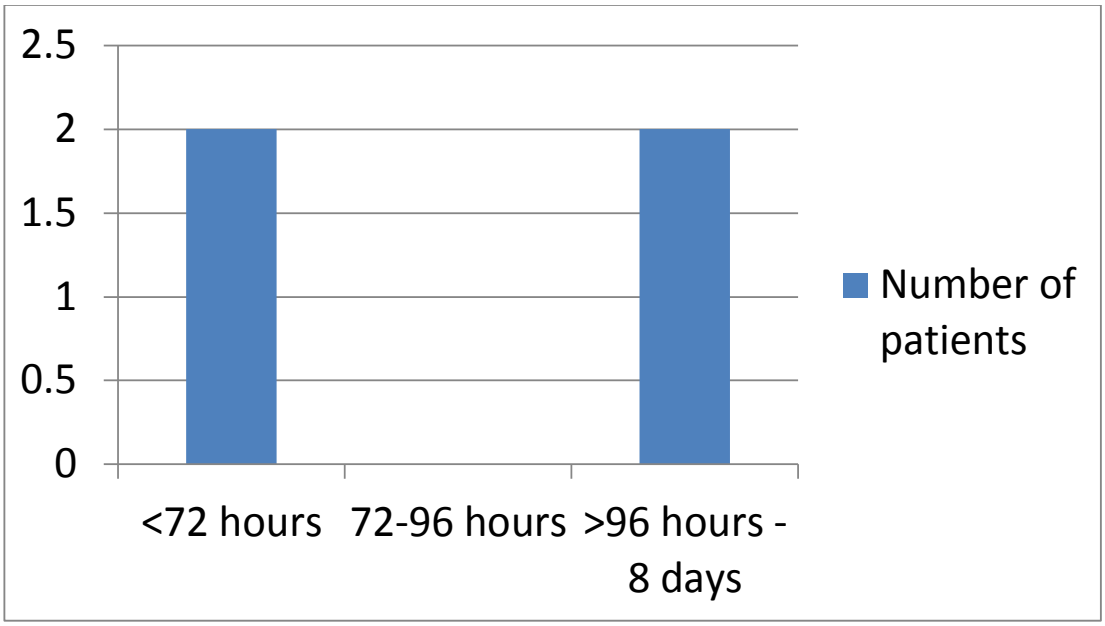
GRADE 1 CHOLECYSTITIS




In our study,most of the surgical candidates of Grade 1 disease were of younger age group.

TIMING OF SURGERY IN

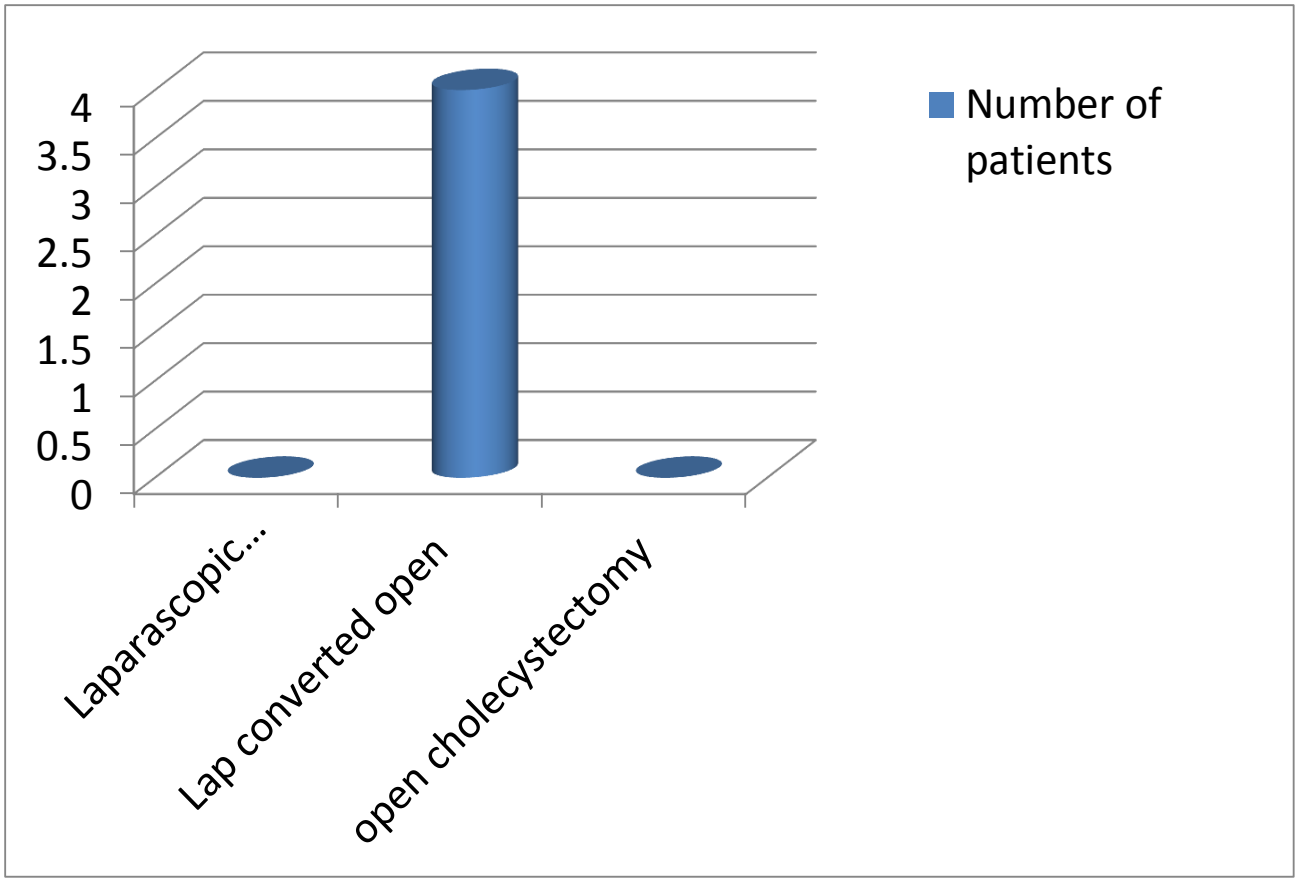
GRADE 1 CHOLECYSTITIS PATIENTS




Only 50 % of the surgical group were subjected to Early Cholecystectomy within 72 hours of presentation as dictated by the TG 13 guidelines.

TYPE OF SURGERY

GRADE 1 CHOLECYSTITIS PATIENTS




All patients in the early surgical group were attempted Laparascopic Cholecystectomy which eventually was converted to open cholecystectomy because of intra-operative difficulty in dissecting the calot's triangle.

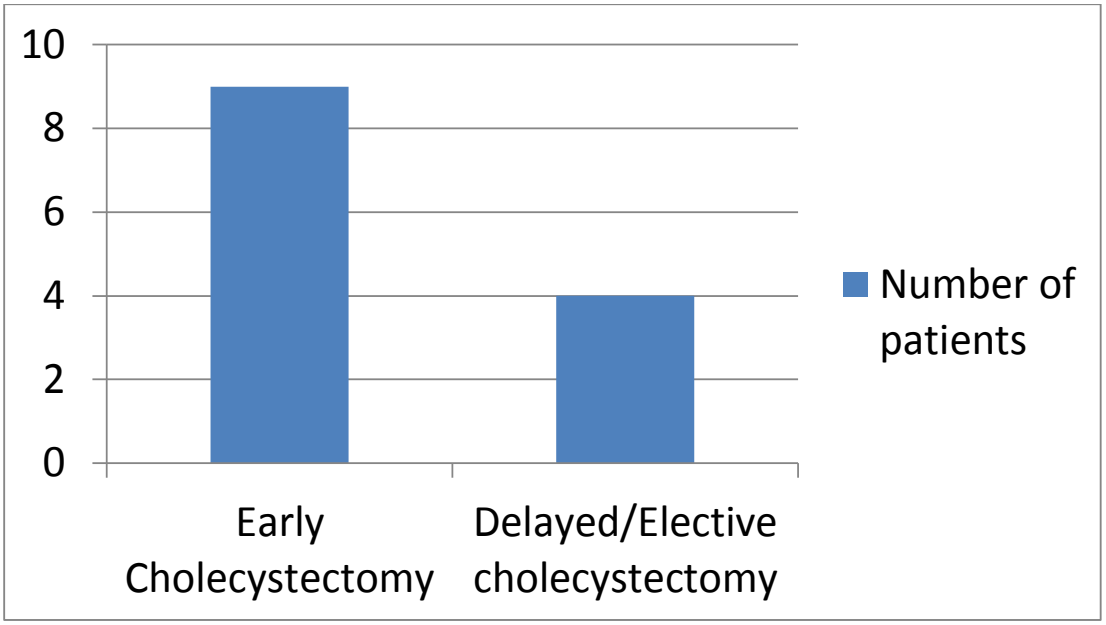
# MANAGEMENT OUTCOMES OF GRADE 2

# ACUTE CHOLECYSTITIS

## TOTAL NUMBER OF PATIENTS(GRADE 2) : 13

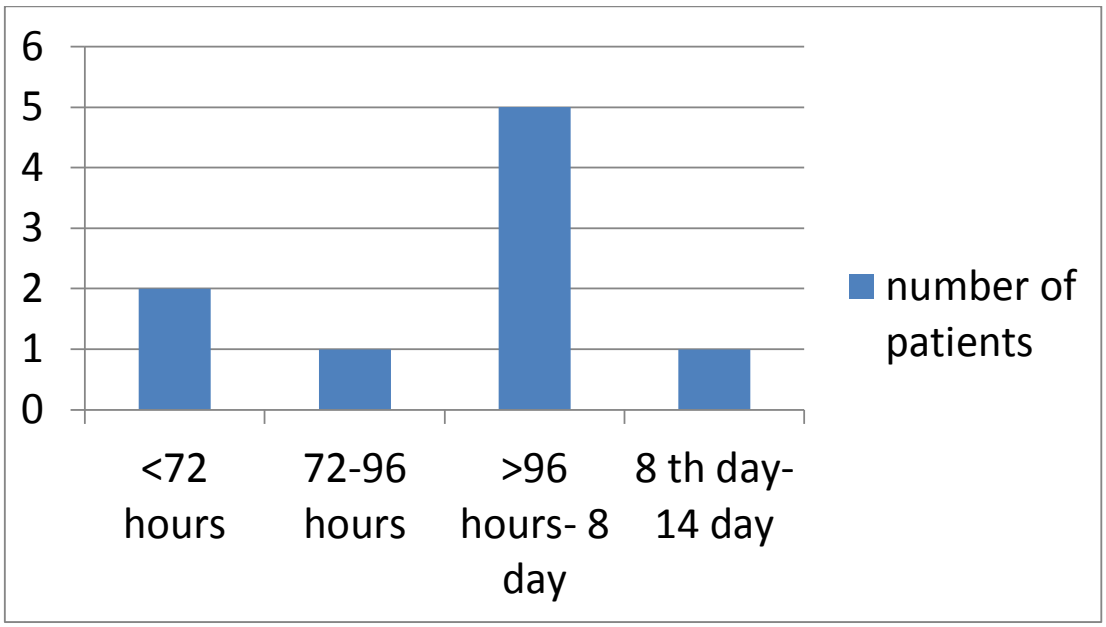
TIMING OF CHOLECYSTECTOMY IN GRADE 2

ACUTE CHOLECYSTITIS PATIENTS




9 out of 13 patients who had grade 2 disease were managed with Early cholecystectomy in our setup .

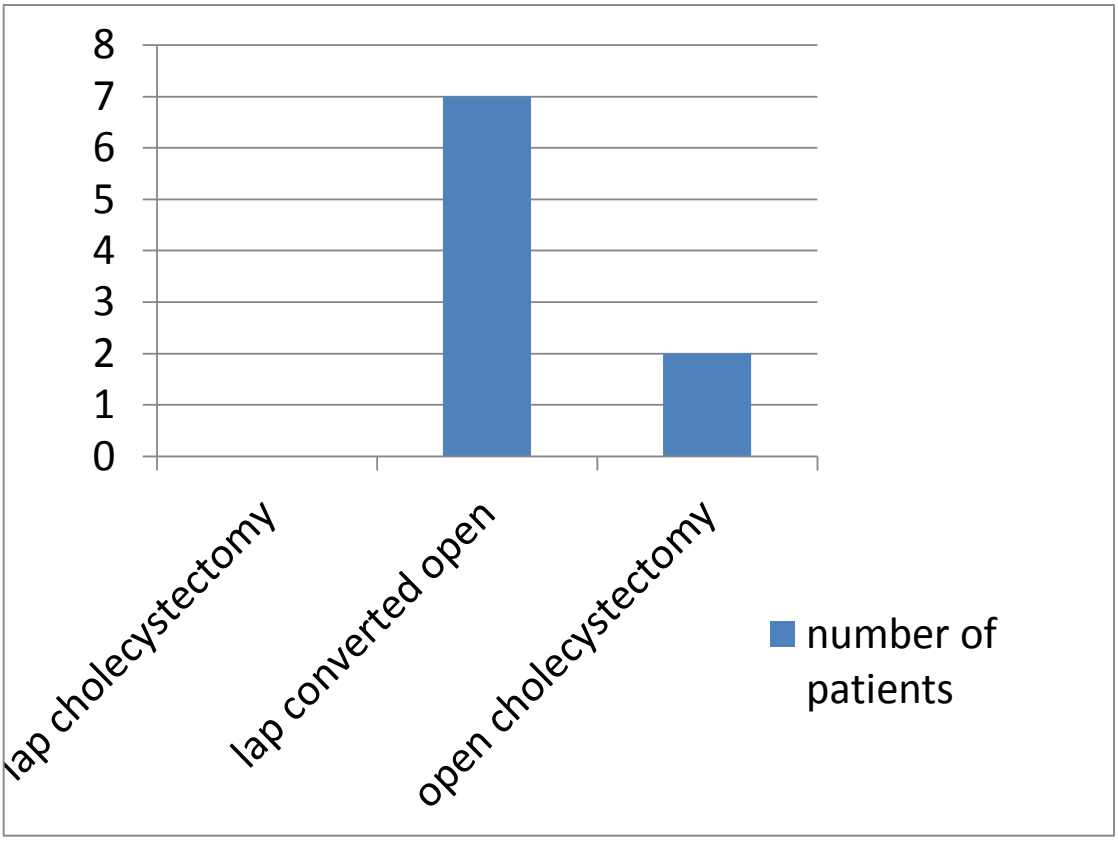
TIMING OF SURGERY IN EARLY SURGICAL GROUP IN GRADE 2 CHOLECYSTITIS




Only 2 patients out of 9 in early cholecystectomy group Had their surgery within 72 hours. Most patients were included in this group had failed Conservative line of management .

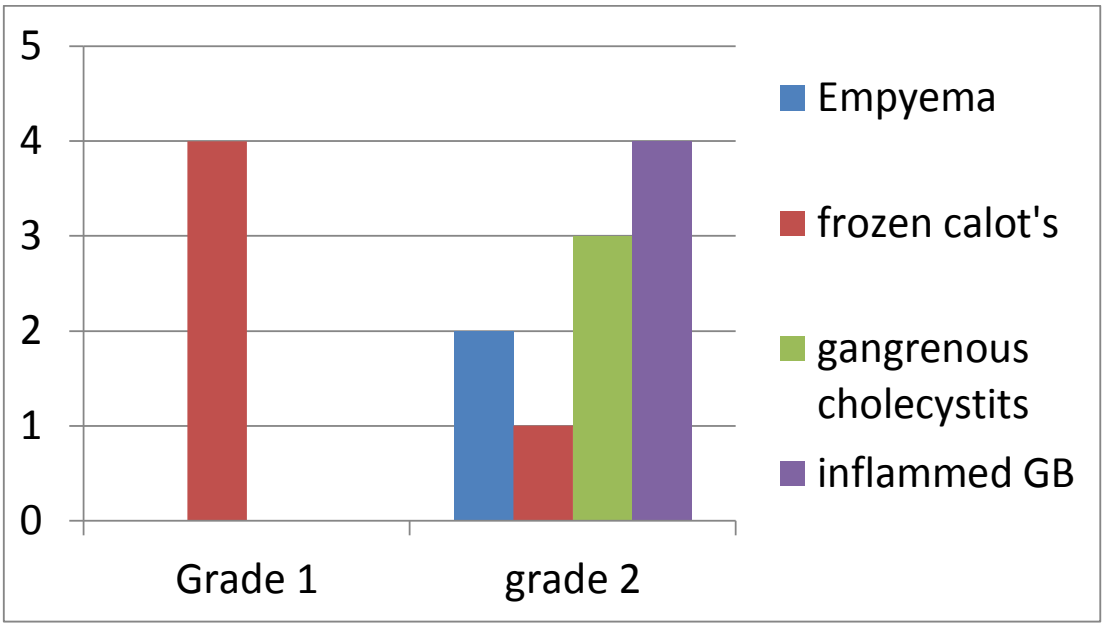
TYPE OF SURGERY

GRADE 2 CHOLECYSTITIS -EARLY SURGICAL GROUP



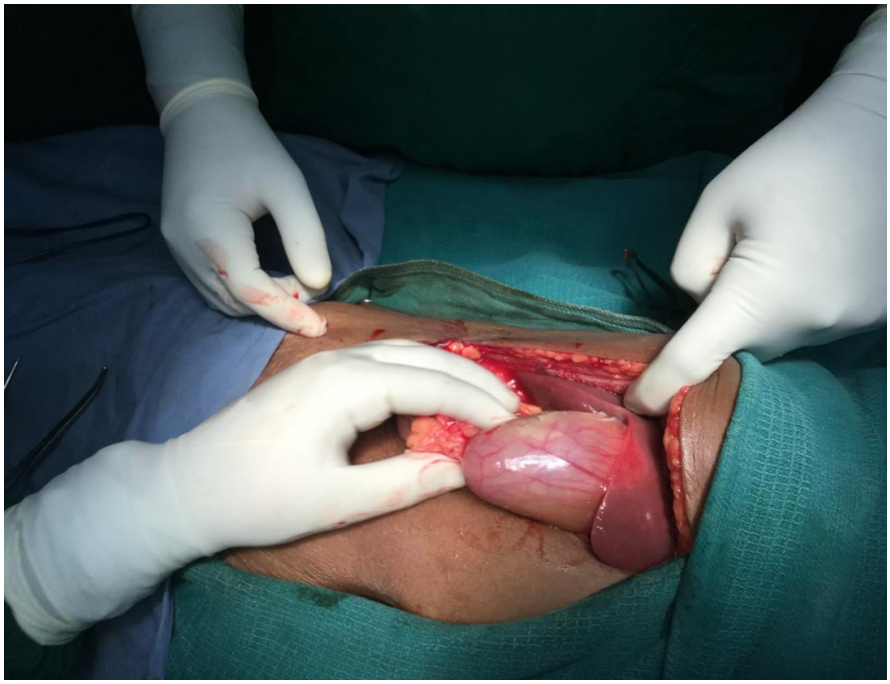

7 out of 9 patients were subjected to laparascopic cholecystectomy which eventually was converted to Open cholecystectomy. None of the patients underwent laparascopic cholecystectomy in Grade 2 disease.

INTRA-OP FINDINGS IN OPERATED GROUP OF PATIENTS



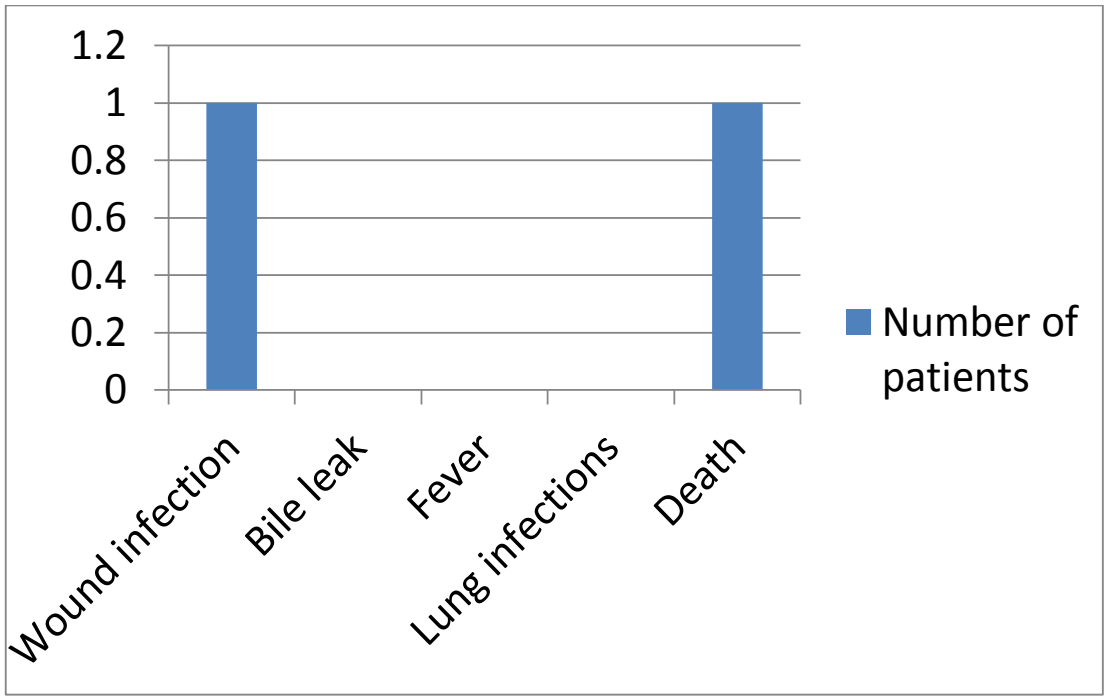

Grade 2 Disease patients were difficult to manage intra-operatively With a myriad of findings intraoperatively.

DISTENDED INFLAMMED GALLBLADDER-INTRA-OP



FROZEN CALOT'S TRIANGLE

POST-OPERATIVE COMPLICATIONS IN OPERATIVE GROUP

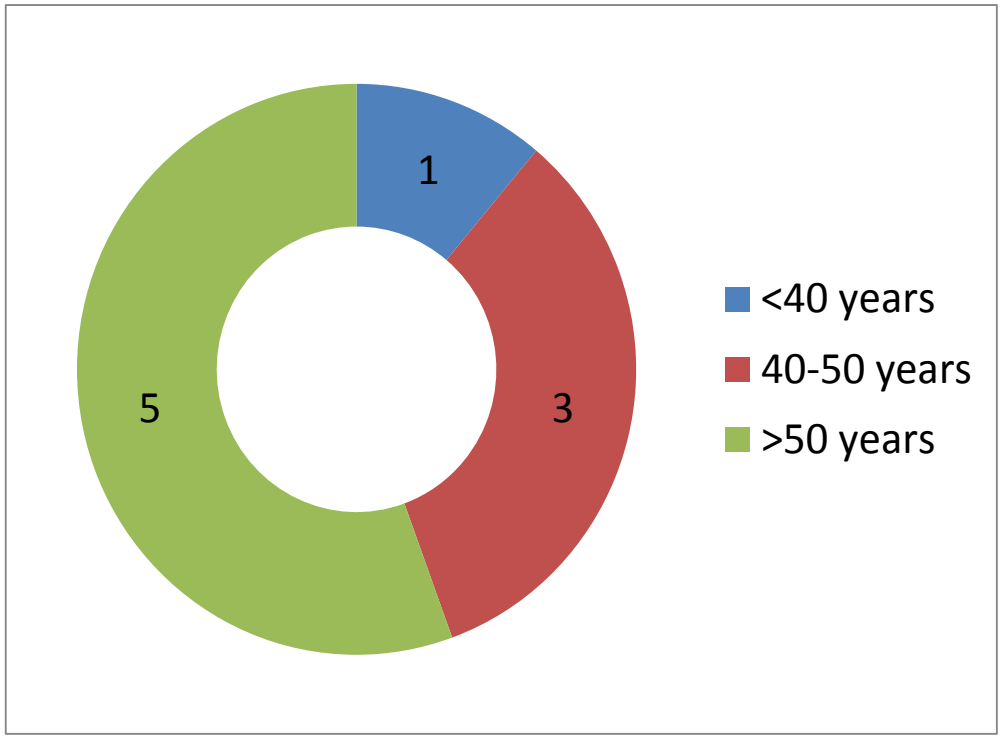



Only one patient had post-operative surgical site Infection.One patient expired of sepsis.

# ANALYSIS OF MANAGEMENT OUTCOMES IN ACUTE CHOLANGITIS

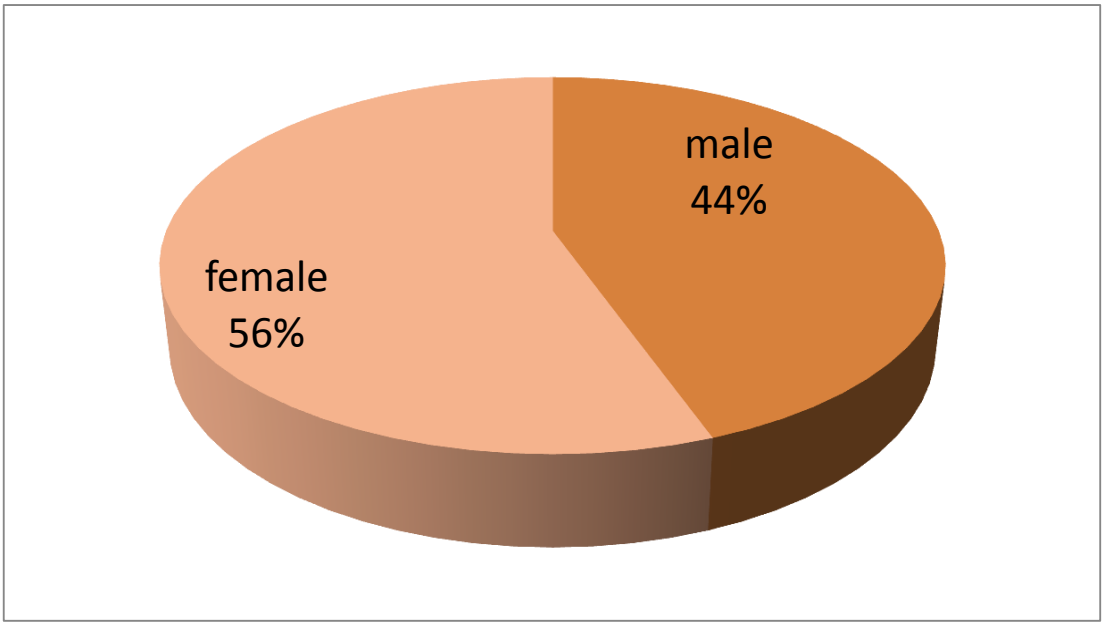
TOTAL NUMBER OF PATIENTS STUDIED : 9

AGE WISE DISTRIBUTION



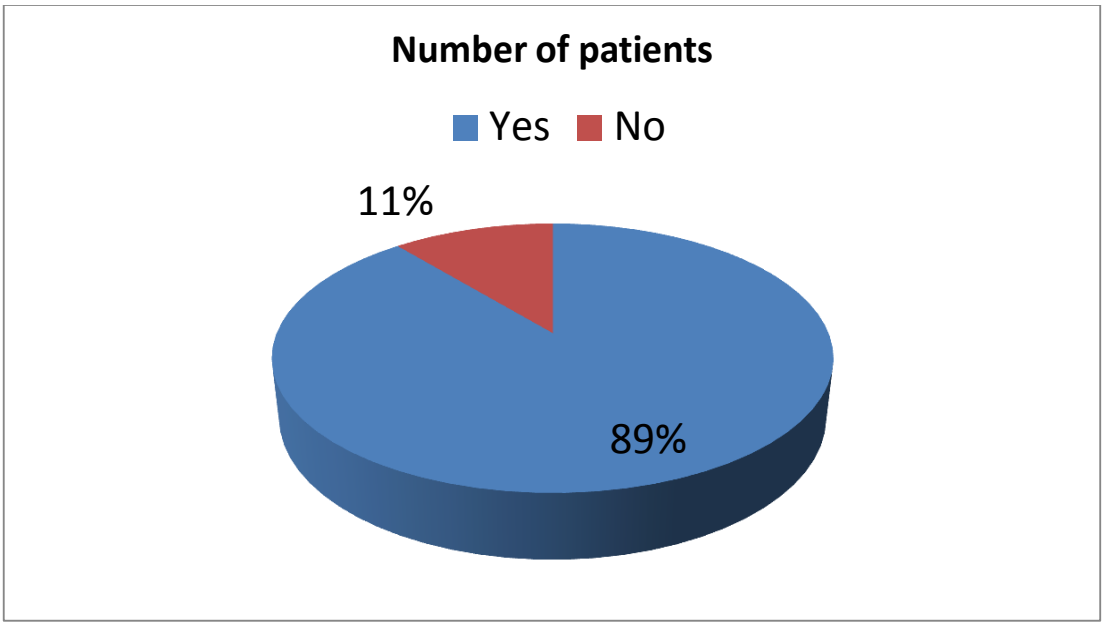

More than 50 % of the study population in the Acute Cholangitis group were > 50 years of age.

SEX DISTRIBUTION



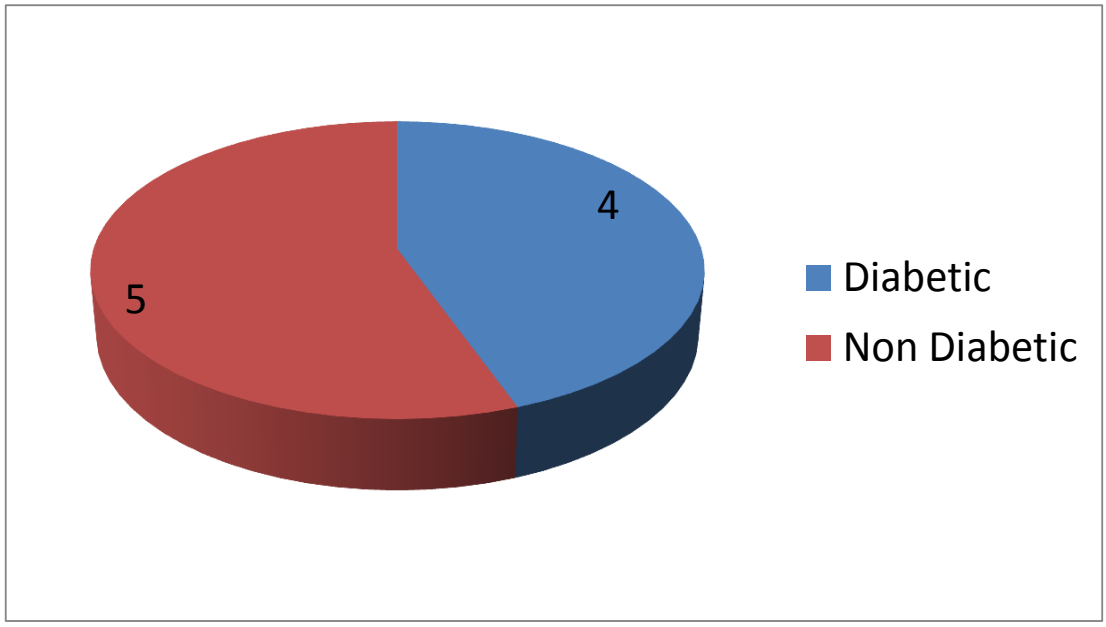

56 % of the cholangitis patients were females in our Study which is on par with literature references.

CHARCOT'S TRIAD



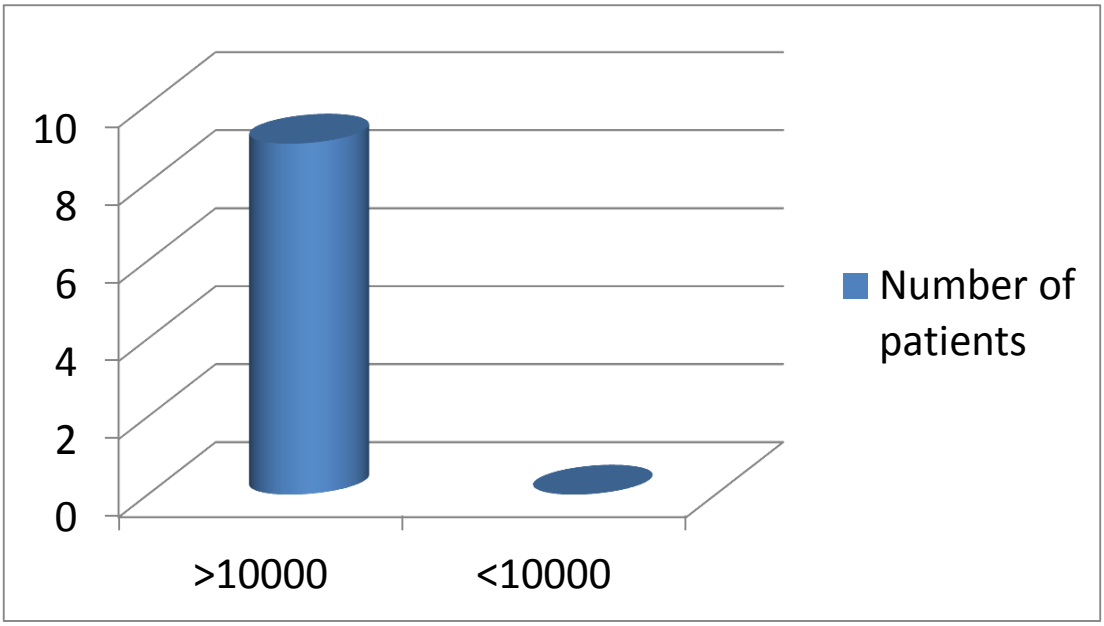

89 % patients in my study group had the characteristic Charcot's triad in Acute Cholangitis.

DIABETES MELLITUS



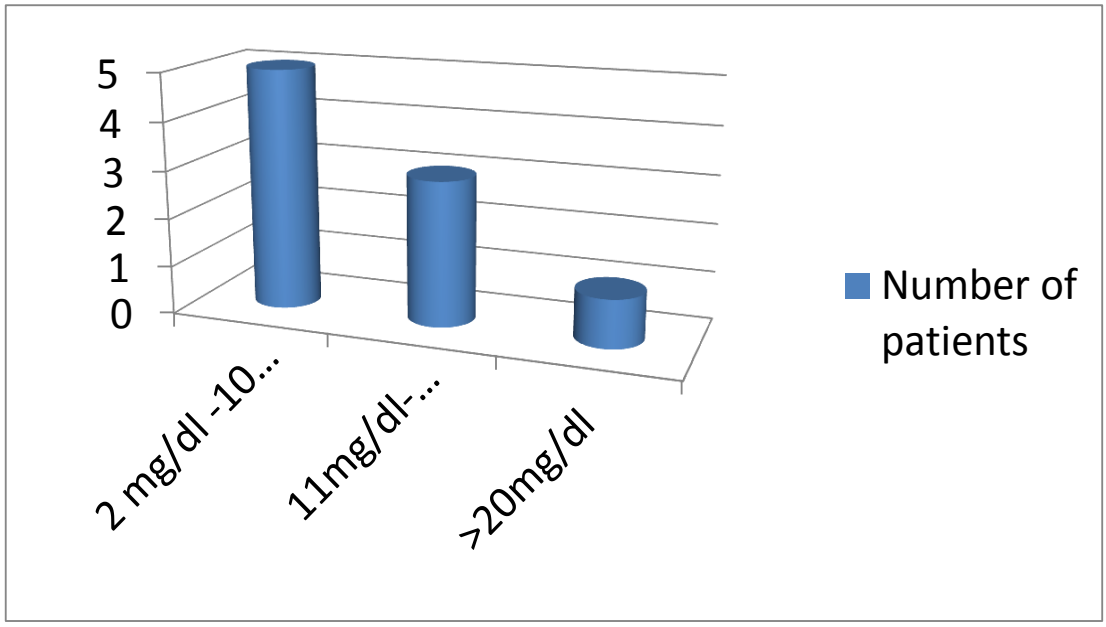

More than 50 % of the patients in my study group were Diabetics and were in some form of treatment for the same

TOTAL LEUCOCYTE COUNT



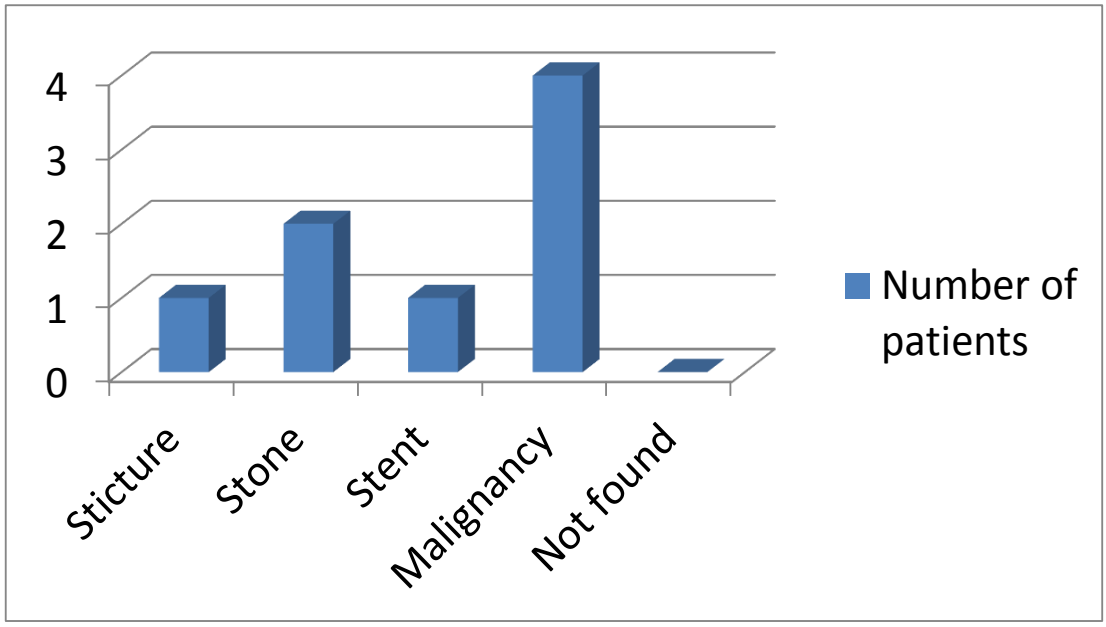

All patients in the study group had an elevated total Leucocyte count of more than 10000 one of the demanding criteria In TG13 guidelines.

TOTAL BILIRUBIN LEVELS



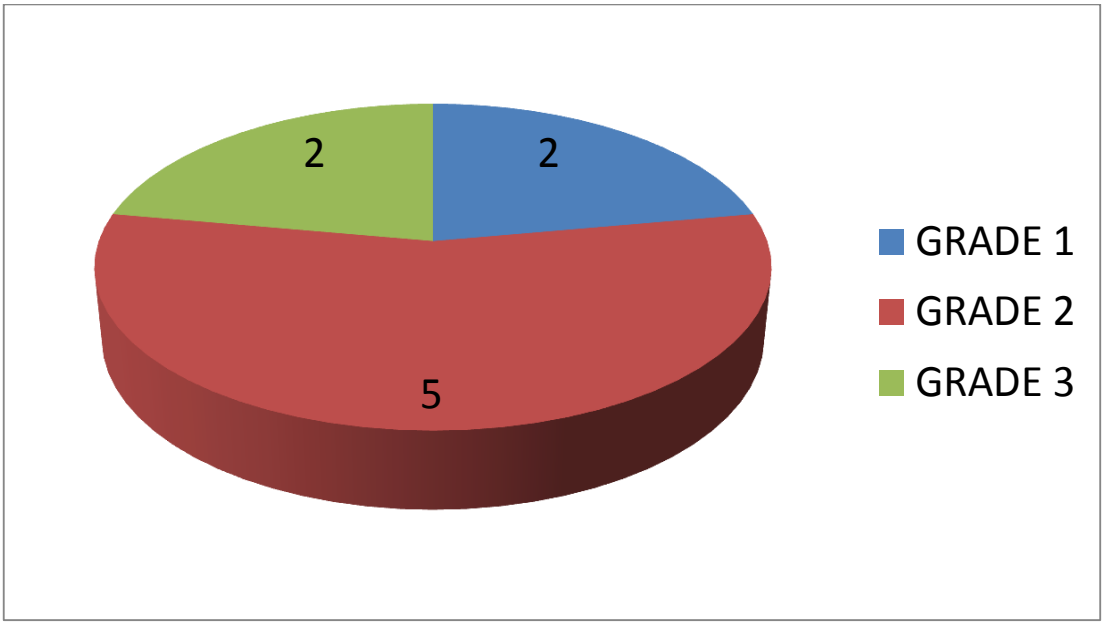

All patients had elevated Total bilirubin levels with most commom range being 2mg/dl -10mg/dl.

CAUSE FOR BILIARY TREE OBSTRUCTION




Malignant obstruction of the biliary tree was the most Common etiological agent.2nd common cause was gallstone Disease.

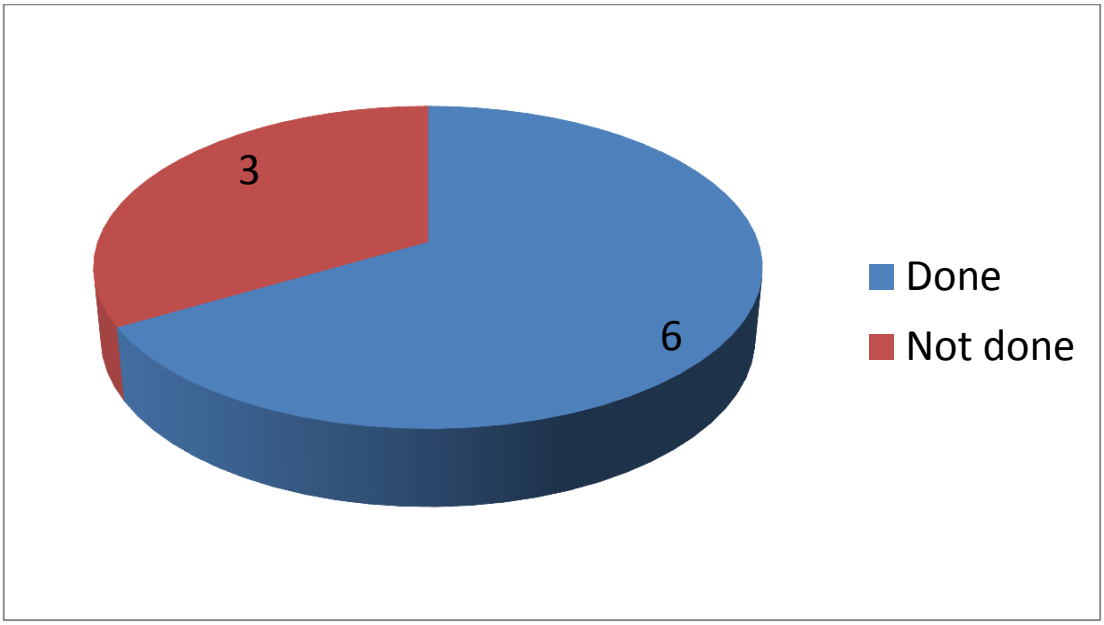
GRADE OF CHOLANGITIS IN STUDY POPULATION




60 % of the patients in my study group had Grade 2 disease or more at the time of Presentation.

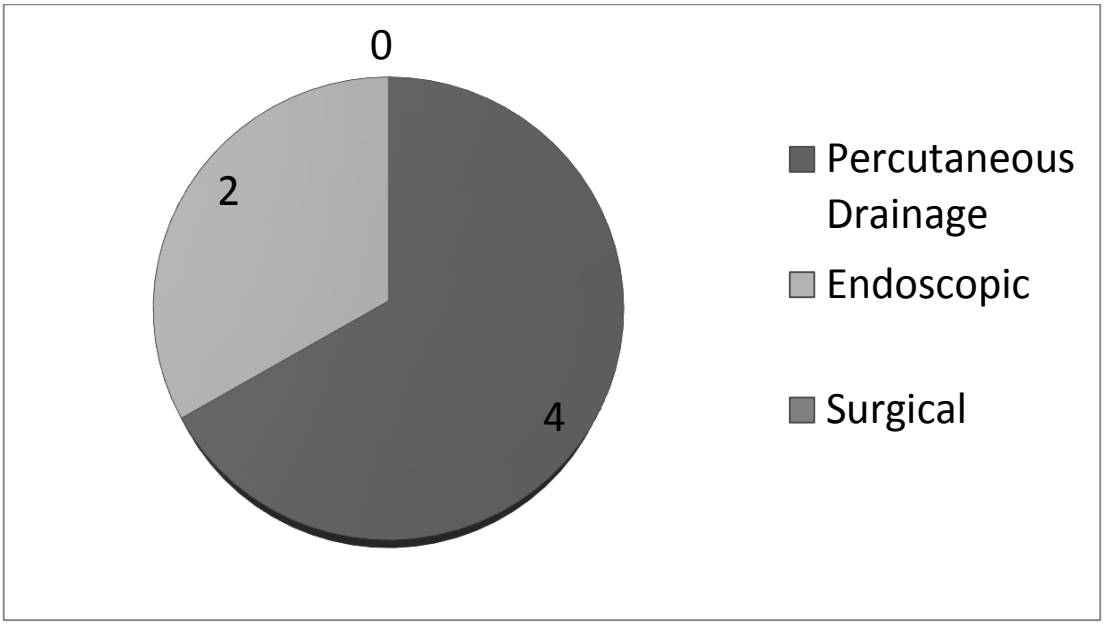
# MANAGEMENT OUTCOMES IN ACUTE CHOLANGITIS GROUP

BILIARY DRAINAGE



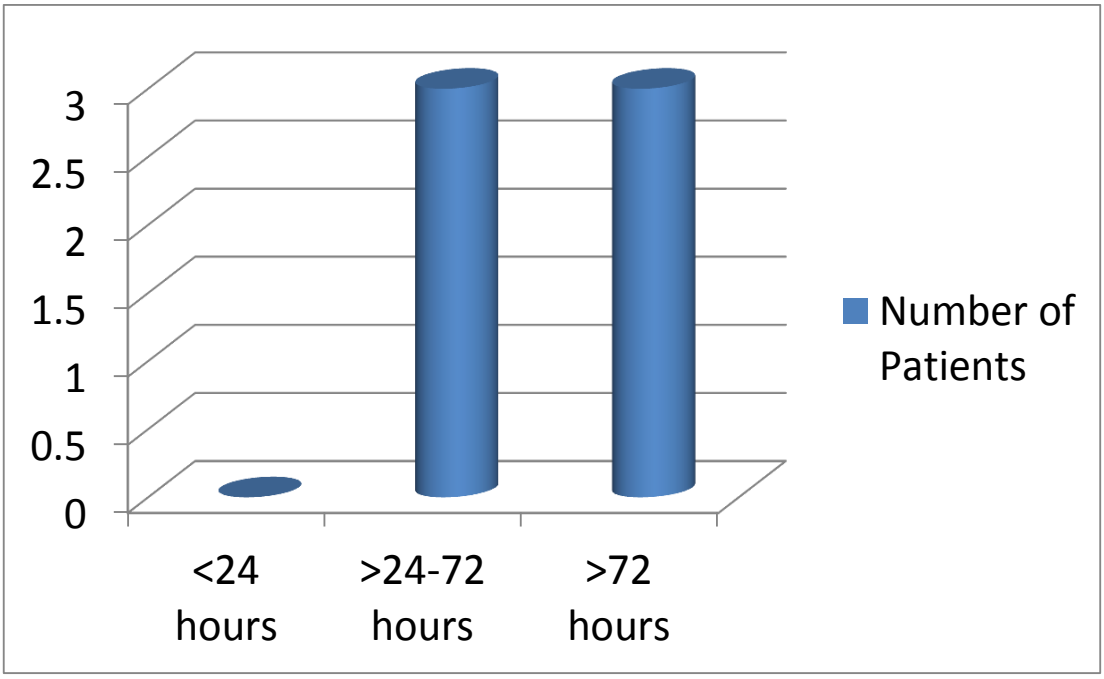

6 out of 9 patients had biliary drainage done after initial stabilisation and recovery.

TYPE OF BILIARY DRAINAGE




Most common Drainage procedure was Percutaneous Biliary drainage . 2 out of 6 patients were endoscopically drained.

TIMING OF BILIARY DRAINAGE FROM DIAGNOSIS




None of the patients among 6 patients who were subjected for biliary tract drainage were drained. In the initial 24 hours.

# DISCUSSION

- Acute Biliary sepsis is more common in elderly patients aged more than 50 years of age.Acute Cholecystitis with most common etiology being gallstone disease is more common in males (56%),While Acute cholangitis is more common among elderly females.(58%)

- Acute cholecystits is most commonly caused by Gallstones,while Acute Cholangitis is most commonly caused by malignant growth causing obstruction to the biliary tract.

- > 52 % patients diagnosed with Acute cholecystitis presented to us only after >72 hours of symptom onset,invariably these patients were categorized as Grade 2 as per TG 13 guidelines

- Acute onset upper abdominal pain was the most common symptom in the study group and tenderness over the right hypochondrium was the most common clinical sign elicited among the study group.

- 43 % of cholecystitis and > 50% cholangitis patients were Diabetics Under treatment for the same.

- Nearly 50 % of the patients in cholecystitis group were afebrile at the time of presentation.

- > 80 % of the study population in cholecystitis group had an elevated leucocyte count of more than 12000 cells/mm 3 and Serum Amylase Levels more than 40 IU/L.

- More than 65 % of the cholecystitis group had a thickened gallbladder wall of size more than 5 mm in the initial screening ultrasound which was the most consistent finding followed by an enlarged gallbladder.

- 57 % of patients with cholecystitis and 60 % of patients with cholangitis Were Grade 2 diseased as per TG 13 severity assessment criteria.

- 60 % of Grade 1 cholecystitis patients were managed conservatively and 70% of Grade 2 cholecystitis patients were managed surgically as the first Modality in the study group.

- 66 % of the cholangitis patients had biliary drainage with percutaneous Route being the most commonly used biliary drainage procedure and Only after 24 hours due to non availability of interventional radiologist and poor general condition.

- 7 out of 9 patients of grade 2 cholecystitis who were managed with early surgery were proceeded with laparoscopic cholecystectomy which eventually got converted to open cholecystectomy in due course due ti intraoperative difficulty.

- Only 2 out of 7 patients in the early surgical group in grade 2 cholecystitis group had their surgery within 72 hours of symptom onset.

- Post-operative complications were minimal in the operated cholecystitis group with wound infection occurring in one patient and one patient died postoperatively because of uncontrolled sepsis despite early treatment.

- CROSS COMPARISONS :

- AGE-SEX IN CHOLECYSTITIS:

  Males aged > 50 years of age had a higher incidence of acute

  cholecystitis

- AGE-SEX-GRADE IN CHOLECYSTITIS:

  Males more than 50 years had a higher grade of disease at the time of presentation.

- DURATION OF DIABETES –SEVERITY OF CHOLECYSTITIS:

  Diabetic Males more than 50 years of age with duration of diabetes less than 5 years had increased incidence of cholecystitis and presented with a higher grade.

# CONCLUSION

- Elderly patients in the 5th decade with diabetes mellitus as co-morbid illness are at high risk for acute biliary sepsis. Routine screening for contributing factors of the disease for these patients may be considered.

- Early laparoscopic cholecystectomy can be considered within 72 hours ,if patient is young and without any co-morbid illness.

- TOKYO GUIDELINES 2013 is an appropriate tool that can be used in our setup without any fallbacks as most of parameters. In the guidelines can be analysed and reproduced in our population group.

- Availability of trained radiologist/intervention radiologist in our Setup needed to be overlooked as early biliary drainage is vital in the outcome of cholangitis and makes us complete in adopting the guidelines.

- A Large multicentre analytical study may be needed to analyse the guideline demanding parameters and other factors that can be added positively as recommendations to the guidelines formulating committee.

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# ANNEXURES

# PROFORMA

**A study on outcomes & efficacy in managing acute cholangitis & acute cholecystitis based on TG 13 Guidelines.**

Investigator: **Dr.S.SIVAPRAGASH** , PG 2nd year – MS (General Surgery)

Guide: **Prof. Dr. LALITHKUMAR,** Chief, Unit S7

- NAME : SL. NO:
- AGE /SEX:
- ADDRESS WITH CONTACT NUMBER:
- IP NO:
- DATE OF ADMISSION:
- DATE OF SURGERY:
- DURATION OF SYMPTOMS:

**HISTORY OF PRESENTING ILLNESS:**

**PAST HISTORY:**

WHETHER A KNOWN CASE OF DM/HYPERTENSION/ASTHMA/TB/EPILEPSY/CARDIAC ILLNESS

H/O SIMILAR EPISODES IN THE PAST, IF ANY:

**CLINICAL EXAMINATION:**

**GENERAL EXAMINATION: TEMP: P.R: B.P: R.R WEIGHT HEIGHT**

**SYSTEMIC EXAMINATION:**

CVS

RS

PER ABDOMEN:

**CLINICAL DIAGNOSIS:**

**BLOOD INVESTIGATIONS:**

| | |
|---|---|
| **HEMAT** | |
| HB | |
| PCV | |
| RBC | |
| TC | |
| DC | |
| PLT | |
| ESR | |
| RBS | |
| CRP | |
| **B.UREA** | |
| **S.CREAT** | |
| **S.Na+** | |
| **S.K+** | |
| **S.Cl-** | |
| **S.HCO3-** | |

**LIVER FUNCTION TESTS:**

| | |
|---|---|
| T.BILIRUBIN | |
| DIRECT BILIRUBIN | |
| AST | |
| ALT | |
| ALP | |
| S.ALBUMIN | |

| | |
|---|---|
| PT | |
| INR | |

LIPID PROFILE : T.Cholesterol: Triglyceride level:

IMAGING: CHEST X-RAY: ABDOMEN X-RAY:

USG ABDOMEN:

1. ANTIBIOTIC USED :

2. DURATION OF ANTIBIOTICS:

3. **SURGERY**:

   DAY OF SURGERY FROM DIAGNOSIS:

4. **TYPE OF SURGERY** : OPEN CHOLE ☐ LAP CHOLE ☐ LAP ☐ OPEN

INTRA-OP:

5. COMPLICATIONS :

6. OTHER INTERVENTIONS,IF ANY:

7. POST –OPERATIVE PHASE: ICU ☐ DAYS: ☐ FEVER

8. TRANSFUSIONS :

9. DURATION OF STAY IN HOSPITAL :

# MASTER CHART

| IP NO | NAME | AGE | Sex | Duration o | First sympt | Bay of rep | Diabetic His | other il | Duration | Under | Treatm | Clinical findings | Fever | Tach | Baselir | Total leuc | Serum k | Serum | Total Ch | ALP | First imagi | USG FINDI | GB STC |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1654632 | RAVI | 40-50 | Male | >72 HOUR | upper abd | Emergency | non-diabeti | nil | | | | mass in right hypochondrium | Yes | yes | >10 | >12000 | <4Meq/ | <40 | <140 mg | >200 | usg abdom | Thickening | no |
| 1655737 | PALAPA | >50 | Male | 24-72 HOU | upper abd | Emergency | non-diabeti | Cardiac illness | | | | Tenderness in Right hypochondrium | No | yes | >10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Thickening | Yes |
| 1647849 | SYED AME | >50 | Male | >72 HOUR | upper abd | Emergency | Diabetes mellitus | | 6-10 yea | yes | OHA'S | local guarding;Tenderness in Right hypoch | Yes | no | >10 | >12000 | <4Meq/ | >40 | >200 mg | < 120 | usg abdom | Thickening | Yes |
| 1655712 | SAKUNTHA | >50 | Femal | 24-72 HOU | upper abd | Emergency | non-diabeti | Systemic Hypertension | | | | Tenderness in Right hypochondrium | No | yes | <10 | <12000 | >4mEq/ | >40 | 141-200 | >120-2 | usg abdom | Thickening | Yes |
| 1655957 | ABDUL MA | >50 | Male | >72 HOUR | fever | Emergency | Diabetes mellitus | | 6-10 yea | yes | OHA'S | local guarding;Tenderness in Right hypoch | Yes | yes | >10 | >12000 | <4Meq/ | >40 | >200 mg | >120-2 | usg abdom | Enlarged g | Yes |
| 1639399 | REKHA | <40 | Femal | 24-72 HOU | upper abd | OPD | non-diabeti | nil | | | | local guarding;Tenderness in Right hypoch | No | yes | <10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Enlarged g | no |
| 1611234 | KALAIARAS | >50 | Femal | 24-72 HOU | fever | Emergency | non-diabeti | nil | | | | Murphy's sign;Tenderness in Right hypoch | Yes | yes | <10 | >12000 | >4mEq/ | >40 | >200 mg | >200 | usg abdom | Thickening | no |
| 1648340 | SHANKAR | >50 | Male | >72 HOUR | fever | Emergency | non-diabeti | Cardiac illness | | | | Murphy's sign;Tenderness in Right hypoch | Yes | no | >10 | >12000 | <4Meq/ | >40 | 141-200 | >200 | usg abdom | Thickening | Yes |
| 1648958 | BABU | 40-50 | Male | >72 HOUR | upper abd | Emergency | non-diabeti | nil | | | | Murphy's sign;Tenderness in Right hypoc | Yes | no | >10 | <12000 | <4Meq/ | >40 | 141-200 | >200 | usg abdom | Thickening | no |
| 1649402 | VEERAPPA | >50 | Male | 24-72 HOU | upper abd | Emergency | Diabetes mellitus | | < 5 years | yes | OHA'S | Murphy's sign;mass in right hypochondriu | No | yes | >10 | >12000 | <4Meq/ | >40 | >200 mg | >200 | usg abdom | Enlarged g | Yes |
| 1639629 | KOMALA | 40-50 | Femal | 24-72 HOU | upper abd | Emergency | Diabetes mellitus | | < 5 years | yes | OHA'S | Tenderness in Right hypochondrium | No | yes | >10 | >12000 | >4mEq/ | >40 | 141-200 | >120-2 | usg abdom | Thickening | Yes |
| 1619056 | MANJULA | <40 | Femal | 24-72 HOU | upper abd | Emergency | non-diabeti | nil | | | | Tenderness in Right hypochondrium | No | yes | >10 | >12000 | <4Meq/ | <40 | 141-200 | < 120 | usg abdom | Thickening | Yes |
| 1514391 | MURUGAV | 40-50 | Male | 24-72 HOU | upper abd | OPD | non-diabeti | nil | | | | Tenderness in Right hypochondrium | No | yes | >10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Thickening | no |
| 1467699 | RADHAKRI | >50 | Male | 24-72 HOU | upper abd | OPD | Diabetes mellitus | | < 5 years | yes | OHA'S | local guarding;Tenderness in Right hypoch | Yes | yes | >10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Thickening | no |
| 1546660 | TAMILSELV | >50 | Femal | >72 HOUR | upper abd | OPD | Diabetes mellitus | | < 5 years | yes | OHA'S | Murphy's sign;Tenderness in Right hypoc | No | yes | >10 | >12000 | >4mEq/ | >40 | >200 mg | < 120 | usg abdom | Thickening | no |
| 1518560 | DHAMODH | <40 | Male | >72 HOUR | upper abd | Emergency | non-diabeti | nil | | | | Tenderness in Right hypochondrium | No | yes | >10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Enlarged g | Yes |
| 1511472 | AZHAGUVE | 40-50 | Male | >72 HOUR | upper abd | OPD | non-diabeti | nil | | | | Tenderness in Right hypochondrium | Yes | no | <10 | >12000 | <4Meq/ | >40 | >200 mg | >120-2 | usg abdom | Thickening | no |
| 1467699 | DAMODAR | >50 | Male | >72 HOUR | upper abd | Emergency | Diabetes mellitus | | < 5 years | yes | OHA'S | local guarding;Tenderness in Right hypoch | Yes | yes | >10 | >12000 | <4Meq/ | >40 | 141-200 | >120-2 | usg abdom | Enlarged g | no |
| 1657934 | Gunasunda | 40-50 | Femal | 24-72 HOU | upper abd | OPD | Diabetes mellitus | | 6-10 yea | yes | INSULI | Tenderness in Right hypochondrium | No | yes | >10 | <12000 | <4Meq/ | <40 | 141-200 | >200 | usg abdom | Thickening | no |
| 1600722 | Palani | >50 | Male | >72 HOUR | upper abd | OPD | non-diabeti | Systemic Hypertension | | | | local guarding;Tenderness in Right hypoch | No | yes | >10 | >12000 | >4mEq/ | >40 | 141-200 | < 120 | usg abdom | Enlarged g | Yes |
| 1658529 | Reshma | <40 | Femal | 24-72 HOU | upper abd | OPD | non-diabeti | nil | | | | Tenderness in Right hypochondrium | Yes | yes | >10 | <12000 | >4mEq/ | >40 | 141-200 | < 120 | usg abdom | Enlarged g | Yes |
| 1653959 | Manohar | >50 | Male | >72 HOUR | upper abd | OPD | Diabetes mellitus | | < 5 years | yes | OHA'S | Tenderness in Right hypochondrium | Yes | yes | >10 | <12000 | <4Meq/ | >40 | >200 mg | >120-2 | usg abdom | Enlarged g | Yes |
| 1636111 | Balaji | >50 | Male | >72 HOUR | upper abd | Emergency | Diabetes mellitus | | < 5 years | yes | OHA'S | Murphy's sign;local guarding;Tenderness | Yes | yes | <10 | >12000 | >4mEq/ | >40 | 141-200 | >200 | usg abdom | Thickening | no |
| | | | | | | | | 9 | | | | | | | | | | | | | | | |
| | | 0 | 15 | 11 | | 9 | 13 | 2 | | | | 10 | 12 | 19 | | 0 | | | | | | | |
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| Number | Locatio | Size of | CT TA | CT Finding | Local Signs | Systemic s | USG FINDI | TG 13 | Mode of M | MANAGEMENT-GRAD | If Early cholecystector | Type of s | intra-operative findi | POST OP COMPLI | Post-oper | Duration o | ANTIBIO | Duration of Antibiotic therapy | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Yes | gb distensi | Right uppe | Fever;Elev | Thickening | Grade 2 | | Delayed/Elective Lap | open cholecystectomy | open cho | empyema of gallbladder | | no | >10 days | piptaz + | 8-10 days | | |
| Single | Neck | >4mm | No | | Right uppe | Elevated W | Thickening | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 4-7days | | |
| Multiple | others | >4mm | No | | Right uppe | Fever;Elev | Thickening | Grade 2 | | Early Laproscopic cho | <72 hours | lap to op | Gangrenous gallblad | WOUND INFECTI | Yes | >10 days | piptaz + | 8-10 days | | |
| Multiple | | | Yes | gb distensi | Right uppe | Elevated C | Thickening | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 8-10 days | | |
| Multiple | | | Yes | visible calc | Right uppe | Fever | Enlarged g | Grade 2 | | Early Laproscopic cho | > 96 hours-8 days | open cho | Gangrenous gallbladder | | Yes | 6-10 days | Ceftriaxo | 4-7days | | |
| | | | No | | Right uppe | Elevated W | Enlarged g | Grade 1 | Surgery | | <72 hours | lap to op | frozen calot's triangle | | no | 6-10 days | Ceftriaxo | 4-7days | | |
| | | | No | | Murphy's s | Fever;Elev | Thickening | Grade 1 | Surgery | | <72 hours | lap to op | frozen calot's triangle | | no | 6-10 days | Ceftriaxo | 4-7days | | |
| Multiple | | | Yes | gb distensi | Murphy's s | Fever;Elev | Thickening | Grade 2 | | Delayed/Elective Lap | open cholecystectomy | | | | | | piptaz + | 4-7days | | |
| | | | Yes | periGB CO | Murphy's s | Fever | Thickening | Grade 2 | | Delayed/Elective Lap | open cholecystectomy | | | | | | Ceftriaxo | 4-7days | | |
| Single | Neck | >4mm | No | | Murphy's s | Elevated W | Enlarged g | Grade 2 | | Delayed/Elective Lap | open cholecystectomy | | | | | | Ceftriaxo | 4-7days | | |
| Single | Neck | >4mm | No | | Murphy's s | Elevated W | Thickening | Grade 1 | Surgery | | > 96 hours-8 days | lap to op | frozen calot's triangle | | no | >10 days | Ceftriaxo | 8-10 days | | |
| Single | Neck | >4mm | No | | Right uppe | Elevated W | Thickening | Grade 1 | Surgery | | > 96 hours-8 days | lap to op | frozen calot's triangle | | no | >10 days | Ceftriaxo | 4-7days | | |
| | | | No | | Right uppe | Elevated W | Thickening | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 4-7days | | |
| | | | Yes | gb distensi | Right uppe | Fever;Elev | Thickening | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 4-7days | | |
| | | | No | | Murphy's s | Elevated W | Thickening | Grade 2 | | Early Laproscopic cho | 72-96 hours | lap to op | empyema of gallbladder | | no | >10 days | Ceftriaxo | 4-7days | | |
| Single | Neck | >4mm | No | | Right uppe | Elevated W | Thickening | Grade 2 | | Early Laproscopic cho | > 96 hours-8 days | lap.chole | non-specific | | no | 6-10 days | Ceftriaxo | 8-10 days | | |
| | | | No | | Right uppe | Elevated W | Thickening | Grade 2 | | Early Laproscopic cho | > 96 hours-8 days | lap.chole | non-specific | | no | >10 days | Ceftriaxo | 8-10 days | | |
| | | | No | | Right uppe | Fever;Elev | Enlarged g | Grade 2 | | Early Laproscopic cho | > 96 hours-8 days | lap.chole | non-specific | | no | 6-10 days | Ceftriaxo | 4-7days | | |
| | | | No | | Right uppe | Fever | Thickening | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 4-7days | | |
| Multiple | Neck | >4mm | Yes | visible calc | Right uppe | Elevated W | Thickening | Grade 2 | | Early Laproscopic cho | > 96 hours-8 days | lap.chole | non-specific | | no | >10 days | Ceftriaxo | 8-10 days | | |
| Multiple | others | >4mm | No | | Right uppe | Fever | Enlarged g | Grade 1 | Conservative | | | | | | | | Ceftriaxo | 4-7days | | |
| Multiple | | | Yes | gb distensi | Right uppe | Fever | Enlarged g | Grade 2 | | Early Laproscopic cho | 8-14 days | lap to op | frozen calot's triangle | | no | >10 days | Ceftriaxo | 8-10 days | | |
| | | | No | | Murphy's s | Fever;Elev | Thickening | Grade 2 | | Early Laproscopic cho | <72 hours | lap to op | Gangrenous gallblad | Death | Yes | >10 days | piptaz + | >10 days | | |
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